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Original Articles and Clinical Cases.

ON DISTURBANCES OF SENSATION WITH ESPECIAL REFERENCE TO THE PAIN OF VISCERAL DISEASE.

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Part III.—Pain in Diseases of the Heart and Lungs.

Introduction.

In my previous papers I have dealt with referred pain and superficial tenderness from the topographical standpoint. Where a single area on the body or on the scalp was well marked out, I laid stress on that particular case, oblivious of whether it was typical of the disease which caused it or not. When an organ was associated with a large number of segmental areas, no attempt was made to show the significance of different groupings of these areas in the diseases of that organ.

The following paper treats, not of the topography of the segmental areas, but of the pain caused by diseases of various organs. Thus I lay no weight on the appearance of a single area on the body or the scalp, but attempt to show the meaning of groups of areas of superficial tenderness.

I do not presume to write a treatise on diseases of the heart and lungs, but only to show how diseases of these organs illustrate the distribution and the origin of referred pain and superficial tenderness.

This enquiry is essentially a physiological one with clinical cases in the place of laboratory experiments. Thus many of the cases I shall give as illustrations are not intended to illustrate the natural history of the disease, but simply serve as links in an investigation of the causes which lead to referred pain.

It has been the custom to speak of many of these pains as "intercostal neuralgia," "rheumatic pains," and by other names of a like kind. The referred headaches associated with these pains have been called "megrim," "cardiac headaches," and the like. I hope I shall be able to show that such words are unnecessary and act perniciously by soothing the conscience of the observer.

CHAPTER I.

Pain in Diseases of the Heart.

§ 1. *Introduction.*

The occurrence and significance of pain during the course of diseases of the heart has not been systematically dealt with in any of the text books on the subject. Most authors mention certain specific instances, such as the pains associated with aneurism, and those of angina pectoris, but no systematic attempts have been made to note the presence or the absence of pain and the character it assumes in each of the types of cardiac disease.

It is useless to select only those cases which suffer from pain, for the absence of pain is often more instructive than its presence. The presence or absence of pain should be noted in each case as part of the routine examination.

Then we must determine whether if pain be present it is local or referred. In some cases, as when it involves the neck or the arm, it is obviously referred; but in many cases it would be impossible to say, had we only the patient's description to guide us, whether it were referred or not. This is especially the case with those pains which lie in the region of the heart itself.

It, therefore, becomes necessary to examine the superficial structures of the chest, back, and arm to see if they be tender or not. For if superficial tenderness is present the pain which accompanies such tenderness is certainly referred.

Such hyperæsthesia of the skin has long been recognised as an accompaniment of affections of the heart. Thus Hilton,[1] mentions tenderness of the upper part of the chest as a symptom in diseases of the heart and great vessels, and Walshe places it amongst the occasional concomitants of cardiac disease. The first of these observers shows throughout his works how close he was to the truth with regard to referred pain and its accompanying tenderness; but he was misled by his knowledge of the gross anatomy of the peripheral nerves and by the want of that knowledge of the construction of his sympathetic system which we owe entirely to Gaskell's initiative. Walshe, with his splendid powers of observation, evidently suspected that hyperæsthesia might be a sympton of importance; for he does not dismiss it lightly, but notes that its significance is a matter for further research. Since then writers on cardiac disease have entirely neglected superficial tenderness, dismissing it with a slighting reference to hysteria under complete misapprehension of Charcot's teaching as to the nature of his hysterical points.[2]

Referred pain and superficial tenderness must remain inexplicable so long as we classify cardiac disease according to the name of the valvular defect. For the chief factor in the production of referred pain and its attendant phenomena is some alteration in the tension within the cavities of the heart secondary to the disease of the valves.

Now in some cases of valvular disease the secondary changes within the cavities of the heart are plain; but in many other cases we are ignorant of the conditions within

[1] "Rest and Pain" [4th edition], p. 258.

[2] Since Mackenzie [*Med. Chronicle*, 1892] and myself independently recognised the significance of superficial tenderness in visceral disease, I only know of the following papers bearing on the occurrence of this symptom in cardiac disease—Mackenzie "Heart Pain" [*Lancet*, Jan. 5, 1895], James [*Brit. Med. Jour.*, June 29, 1895].

the heart under which the circulation is carried on. Our knowledge of these conditions is based on our interpretation of physical signs, and it is in this very interpretation of the same physical signs that the greatest differences of opinion exist. No new physical sign or method of examination has been discovered, and yet our knowledge of the physical signs which point to mitral stenosis has advanced considerably of late. Thus it may happen in the future that the laborious association of certain symptoms with what we suppose to be some one or other condition within the heart may become valueless, owing to an increase in our knowledge of the significance of some particular association of physical signs that has as yet escaped observation.

I shall, therefore, in all cases state the conditions which determine the presence or absence of referred pain in terms of physical signs. Later, I shall devote a chapter to a consideration of what I believe to be the interpretation of these signs, and the bearing of this interpretation on the causes of referred pain. Thus if my interpretation should prove incorrect, I hope that the observed association between the presence or absence of pain and certain conjunctions of physical signs may still remain.

Every case upon which this work is founded has been taken by myself. For in such an investigation it is all important that the reader should have to deal with the errors of a palpable individual, rather than with those of impalpable ghosts. In every case from the Victoria Park Hospital for Diseases of the Chest, the physical signs were checked by other observers more skilled than myself. To Dr. Harrington Sainsbury and Dr. Colbeck, my thanks are particularly due for the trouble they took to insure the accuracy of my physical signs in case of cardiac disease.

My object here is to elucidate the conditions which cause referred pain, and I have therefore excluded all cases from these pages in which competent observers diagnosed compound lesions. For although such compound lesions form very instructive puzzles for a commentary, they can only be explained when we have settled the primary laws by a study of simple lesions.

§ 2. *Local Pain in Diseases of the Heart and Pericardium.*

The pain that is produced by diseases of the heart may be local or referred. Before passing to the characteristics and distribution of referred pain, which forms the most important portion of this chapter, I will touch shortly on some features of the local pain that occurs with certain cardiac lesions.

In examination of every patient it is necessary first to exclude referred pain, and its accompanying superficial tenderness before concluding that the pain is local. Now when the pain is situated over the arm, no difficulty can arise, for such pain in disease of the valves of the heart must be referred and not local. But when the pain lies, at any rate, partly, over the heart itself, such pain is commonly, though in most cases erroneously, supposed to be local and not referred.

Attention to the following points of difference enable us to decide whether the pain be local or referred :—

Referred Pain.	Local Pain.
1. Referred pain is not only situated on some spot on the front of the chest, but also over some spot behind at a distance from the cardiac area.	1. Local pain is confined strictly to the area of the heart or pericardium, and does not pass round the body or through to the back.
2. Referred pain is associated with more or less tenderness of the coverings of the chest and back, elicited by picking up the skin and subcutaneous tissues between the fingers.	2. Local pain is accompanied by no marked superficial tenderness. But when the intercostal spaces over the painful area are either pressed or percussed, the pain is increased, the amount of tenderness elicited depending on the force applied.
3. This superficial tenderness frequently passes round the body in a more or less horizontal band from the middle line of the back to the middle line in front. If less acute it lies over one or more spots, one of which is situated over the anterior, the other over the posterior aspect of the trunk.	3. When present, deep tenderness can only be elicited over the heart or pericardium, and is absent from the posterior aspect of the trunk.
4. Pressure as a rule relieves superficial tenderness.	4. Pressure increases deep tenderness.
5. The superficial reflexes are increased over an area of the superficial tenderness accompanying referred pain.	5. The superficial reflexes are unaltered over the area of deep tenderness accompanying local pain.
6. Referred pain is frequently accompanied by headache and superficial tenderness of the scalp, obeying rules with regard to its distribution which I laid down in my second paper.	6. Local pain is unaccompanied by that form of headache which is associated with scalp tenderness. Headache if present is a separate phenomenon, standing in no direct relation to the local pain and deep tenderness.

A.—*Local Pains in Valvular Diseases.*

It is commonly supposed that local pain is far commoner in cardiac disease than referred pain. This I believe to be a pure assumption. Probably local pain is a distinct feature of the majority of painful cardiac states, but it is usually so masked by referred pain that its presence is very difficult to determine.

But in certain cases of mitral disease, especially when the pulse is irregular, the patient complains of palpitation accompanied by pain. This pain is said to lie over the apex beat, and is unaccompanied by superficial tenderness. It seems to be a true local pain. Now this element must exist in other cases in which referred pain is also present; but the presence of this referred pain masks the local pain, and to say that a true local pain is present is but an assumption.

In the same way, in anginal attacks of the suffocative type [*vide* §7], an initial, stretching, bursting pain is felt at the epigastrium, which is undoubtedly local, and is a phenomenon quite apart from the referred pain. Local pain seems to be most often present when the heart is acting irregularly, and is peculiarly associated with a feeling of breathlessness.

Such local pain in the heart is not, however, definitely associated with deep tenderness, but simply lacks all the points which characterise a referred pain.

B.—*Pain in Pericarditis.*

When the pericardium is acutely inflamed, pain is present, which shows the most striking contrast to referred pain. Like inflammation of the pleura and peritoneum, inflammation of the pericardium produces a true local pain, with more or less marked deep tenderness.

But in the adult, inflammation of the pericardium is frequently associated with marked disease of the endocardium. Thus in adults the true phenomena due to inflammation of the pericardium are frequently masked, and all satisfactory examination rendered impossible by the coincident superficial tenderness due to the endocarditis.

Occasionally, however, the opportunity arises of examining a case in which superficial tenderness is absent. The local character of the pericardial pain and its association with tenderness on pressure and percussion (in the interspaces) are then extremely manifest. It is also interesting to see how closely the area of deep tenderness corresponds in some cases with the area over which friction is audible. Case No. 1 shows all these points distinctly, and happened to be one in which all the characteristics of the local pain were demonstrated to the satisfaction of several other observers.

Case 1.—*Illustrating the local pain and deep tenderness produced by Pericarditis.*—Nellie T. [Marylebone Infirmary, Mr. Lunn], aged 15, servant.

November 16, 1895.—The left knee began to swell and was very painful. She remained in bed two weeks as the doctor said she had rheumatic fever. Towards the end of the two weeks her heart became bad, and she was admitted December 2, 1895.

No previous attacks of rheumatism. Nothing worthy of note in the past or family history.

On admission [*December* 2, 1895].—The face was pale, with a slight underlying grey look, but no actual cyanosis. No œdema. No jaundice. The right wrist was swollen and painful, but the fingers were unaffected. The left wrist is becoming swollen.

Respiration is rapid with evident subjective dyspnœa. The alae nasi are dilating and trembling irregularly. Short, dry, hacking, semi-voluntary cough. Speech interrupted by the shortness of breath. Chest moves with every inspiration, and the respiratory rate is about 50 in the minute.

She complained of pain over the heart, and this pain was much worse when she coughed. The pain lay definitely over an area in the fourth and fifth spaces on the left side, about the size of the palm of the hand. The pain did not go through to the back or radiate round the body. There was no pain elsewhere, and no headache.

The most careful examination failed to reveal any superficial tenderness anywhere. She is a most intelligent girl, clever, and bright beyond her years.

On the other hand, there was exquisitely marked tenderness on pressure and on percussion over an area in the fourth and fifth spaces, about the size of the palm of the hand, and with its centre about in the left nipple line.

Cardiac pulsation is widely visible in the fourth and fifth spaces, and also at the epigastrium.

Cardiac dulness begins above at the upper border of the second rib, extends $1\frac{3}{4}$ in. [4·5 cm.] to the right of the middle line, and 5 in. [12·5 cm.] if not more, to the left of the middle line.

Rough to and fro friction is heard in the fourth and fifth intercostal spaces over an area corresponding very closely to that of the tenderness on precussion and pressure.

Over the apex, and as far inwards as the left sternal border, the first sound is absent, and is replaced by a soft blowing systolic murmur which is not conducted into the axilla. Over the lower part of the sternum, both first and second sounds are audible.

Pulse 124, regular, low tension, compressible with distinct tendency to dicrotism.

No capillary pulsation. Pulsation in the veins of the neck.

Beyond some few crackling sounds at the left base, no abnormal physical signs present in the lungs.

Temperature on admission 102. Fell next day to 100·2.

Urine, specific gravity 1020, acid. No albumen.

Three leeches were applied over the area of deep tenderness. The ease they gave was obvious, and, within five minutes from the time they bit, she said she no longer felt the pain, and that her breathing was easier.

In four days all friction had disappeared, but the signs of fluid in the pericardium were exceedingly marked. The cardiac dulness extended considerably more to the right, and as high as the junction of the first rib, with the sternum on either side. The deep tenderness disappeared, and never reappeared throughout the course of the case.

§ 3. *Referred Pain in Diseases of the Aortic Valves.*

I have begun with the pain which appears with lesions of the aortic valves, because from its position over the upper part of the chest and arm, at a distance from the heart, it is most obviously referred. Again, the association of the presence or absence of pain with certain conjunctions of physical signs is striking. And, lastly, the interpretation of these signs is less open to dispute than is the case with lesions of other valves.

Pain of the character I am about to describe, occurs most typically in young people who, as a sequel to acute rheumatism, present the following typical signs: a diastolic murmur over the sternum, conducted more or less towards the apex beat, accompanied by a systolic murmur over the upper part of the chest, conducted into the great vessels, and absence of the aortic second sound. The first sound is present over the apex beat or in the axilla, and no mitral systolic murmur is audible at the angle of the left scapula.

But sometimes an attack of acute rheumatism leaves behind it a loud systolic murmur, heard over the aortic area, followed by a perfect second sound and no diastolic murmur of any kind. The pulse is peculiar neither for the strong stroke and low tension, characteristic of regurgitation, nor for the small stroke and maintained tension which accompanies marked stenosis. Such cases present no marked symptoms, and pain is absent. The lesion of the aortic valves, though acoustically patent, is probably physiologically of little importance.

Pain is also a frequent symptom in those cases of aortic regurgitation which arise without a history of acute rheumatism in men of middle age and laborious occupation. But here again the aortic second sound is absent. No mitral systolic murmur must be audible, and the pulse must be collapsing in character.

When present the pain tends to be situated over the upper three or four intercostal spaces of the left half of the chest, in the upper interscapular region on the left side of the back, and on the inner side of the left arm as far as the elbow. Pain is also sometimes present in the left infra-clavicular region, and over the left side of the neck. This pain is frequently present on the left side alone, but if it is severe it spreads according to the laws I laid down in my first paper, and involves the same parts on the right side of the chest and back, and the inner side of the right arm. In such cases the pain appears later on the right side than on the left, and disappears from the right side earlier than from the left half of the chest.

When severe the pain is said to shoot and dart, but when less severe it is stationary over certain points. Thus the pain on the inner side of the arm to the elbow, when severe, is said as a rule, to shoot upwards from the elbow to the shoulder, but when less severe to be situated over the inner condyle of the humerus.

If the pain in the chest is severe the patient not uncommonly complains of headache, "like neuralgia," situated in the forehead, "over the nose and eyes." This headache comes and goes with the pain in the chest. It is

more marked when the pains in the chest and arm are severe. It is more marked on the left side of the forehead than the right, and is sometimes confined to the left side of the head, in cases where the pain is confined to the left side of the chest and arm. The eyes are said to ache when the headache is present, and not uncommonly the patient becomes unable to read, owing to the pain in the eyes, even when no error of refraction is present. If, however, latent hypermetropia exists, this hypermetropic defect may become manifest under the influence of the headache.

When the sharpness of the pain has somewhat abated, the patient not uncommonly complains that the chest feels sore. On examination, certain areas of the skin of the chest and arm are found to be tender, just as with every other referred pain of sufficient severity and duration. This tenderness of the superficial structures lies mainly over the second, third, and fourth interspaces in front, the inner aspect of the left arm, and an area of the back, situated roughly between the level of the first dorsal and the sixth dorsal spines. The area involved comprises the second, third, and fourth dorsal segmental zones to a greater or less extent. The third and fourth cervical areas are also frequently affected and the pain is then said to extend above the clavicle.

In the same way the headache is associated with distinct tenderness of the superficial structures of the forehead, occupying mainly the frontal, mid-orbital, and fronto-temporal segmental areas of the scalp.

To illustrate these points I give in detail the case of a young girl with marked double aortic murmurs, retention of the faint sound in the left axilla, and no systolic murmur heard at the angle of the left scapula. [Case No. 5, table i., p. 166.] The pain was never very severe, excepting after some particularly indiscreet exertion, to which these aortic cases are peculiarly prone. The tenderness represented [fig. 1] is that usually present when she was not confined to her bed.

Case 5.—*To illustrate the non-paroxysmal referred pain produced by lesions of the aortic valves.*—Louisa I. (Marylebone Infirmary, Dr. Lunn), aged 19, servant.

At the age of 13 she had rheumatic fever, during which the right wrist and left ankle swelled and became painful. She was more or less ill for ten months. About a year afterwards was suddenly taken bad with a pain in the upper part of the left side of the chest and in the neck. In 1892, at the age of 16, she went to service. She then suffered from attacks of shortness of breath, especially after exertion. In 1893 pain became a marked feature, and she was admitted to the infirmary. She much improved, and was discharged, but was re-admitted in 1894. She again improved, and took another situation, but the pain became so bad she was re-admitted in October, 1895.

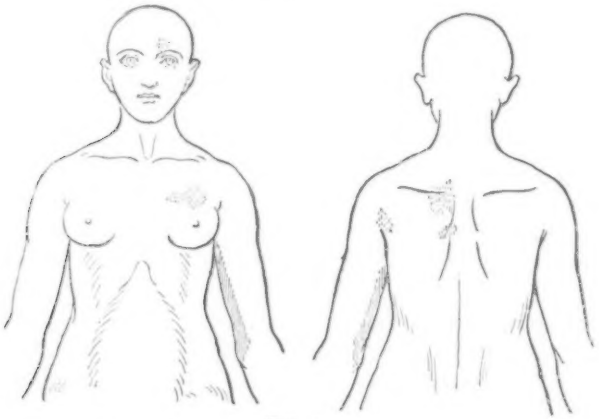


FIG. 1.

To show the areas of superficial tenderness in a Case of Aortic Obstruction and Regurgitation [Case No. 5.]
The area mainly affected is that of the 2nd dorsal segment.
Slight scalp tenderness was present on the left side of the forehead.

December, 1895.—Her condition was as follows:—Very bright, small, slight girl of 19. The greater part of her face is pale, with an irregular underlying vascular mottling. On her cheeks is an irregular pink, mottled flush, which comes and goes. No true anæmia. No œdema. No jaundice. No clubbing of fingers. No nodules. No wasting.

She has no feeling of faintness except with the attacks of pain. When not in bed the heart beats very fast on any exertion, and this seems to bring on the pain. Not short of breath when in bed, but when up and about she has a tight feeling in her chest as if she was going to be choked.

When kept strictly in bed she is free from pain and headache, and all superficial tenderness is absent. When up she is an exceedingly active worker. Pain then appears over the second

left intercostal space, close to the anterior axillary fold, which passes through to the back, close to the vertebral border of the spine of the left scapula. A variable amount of superficial tenderness is present, mainly over the area of the second dorsal segment (fig. 1).

She has occasional headache over the eyes, with slight tenderness of the forehead on the left side (fig. 1).

The pulse, when she is up, is about 100, regular, of good stroke, but typically collapsing. Marked pulsation of the arteries of the neck.

Marked capillary pulsation.

No pulsation of the veins of the neck.

Heart's apex beat in fifth space four inches (10·5 cm.), from the middle line (just outside nipple line). It is heaving. No thrill.

Cardiac dulness beginning at third rib, extends to right edge of sternum, and four and three-quarters inches (12 cm.) to the left (about one inch outside the left nipple line).

Over the whole front of the chest, extending to the mid-axillary line, a loud systolic murmur is heard, followed by a diastolic murmur, forming the typical to-and-fro aortic murmurs. Over the aortic area neither the first nor the second sounds are heard. Over the actual apex beat the first sound is just audible as a dull booming, but on travelling outwards into the left axilla the first sound becomes quite distinct, and is long and booming, in spite of the fact that the aortic systolic murmur is still faintly heard. At the angle of the left scapula a booming first sound is distinctly audible, unaccompanied by any murmur. The pulmonary second sound is audible, but is not accentuated.

Respiration 24, quiet, regular. No cough.

No abnormal physical signs in lungs.

Tongue clean, moist. Appetite good. No pain after food. Bowels irregular.

Urine normal. No albumen.

Vision $\frac{6}{6}$. Jäger 1. No hypermetropia.

Up till April, 1896, the physical signs and main features of the case have remained unaltered.

A marked feature of the pain which appears in aortic disease is the tendency it shows to sudden exacerbations. The pain rapidly spreads, the patient feels exceedingly ill, and, if the spread of the pain is sufficiently rapid and wide, the attack may resemble true primary angina pectoris. Although no hard-and-fast line separates these attacks of increase of pain from the violent anginal attacks which occur in some patients with aortic disease, I shall deal with the whole subject of paroxysmal pain and angina pectoris in a later section [§ 7, p. 202].

All cases of aortic regurgitation, or of regurgitation and stenosis, are liable to suffer from occasional referred pain and tenderness so long as the apical first sound is good and no mitral systolic murmur is heard at the angle of the left scapula. That is to say that so long as the mitral valve holds, pain is liable to appear. If, however, the first sound at the apex and in the axilla is replaced by a systolic murmur conducted to the angle of the left scapula, pain, such as we have learnt to recognise as the possible accompaniment of aortic disease, is absent.

If a patient has long suffered from referred pain and tenderness over the upper dorsal segments, and the mitral valve then fails as a late feature of the disease, it might be said that the pain ceased because the patient's faculties were too low to originate or to appreciate pain.

I have, therefore, collected a series of cases from my notes in which the apical first sound was abolished and replaced by a systolic murmur, conducted to the angle of the left scapula, as a primary feature of the disease. In these cases the mitral regurgitation was not a secondary matter due to heart failure. I have arranged them in a table so that they be compared with a similar table of those cases in which the first sound at the apex was intact, and no systolic murmur was audible at the angle of the left scapula. The absence of referred pain and tenderness in the former group, and its presence in the latter, is striking.

Thus the conclusions to which we have arrived in this section may be summed up as follows :—

(1) When an aortic diastolic murmur, with or without an aortic systolic murmur, is present, the aortic second sound abolished, and, when the pulse shows marked collapsing characteristics, referred pain is liable to form a symptom of the disease.

(2) If in addition to these signs the first sound is absent both over the apex and in the axilla, and a systolic murmur is heard at the angle of the left scapula, referred pain of cardiac origin will be absent. If this condition arises owing to cardiac failure, the pain which was previously present will disappear, and those cases in which this conjunction of

TABLE I.—DISEASE OF THE AORTIC VALVES, WITH A GOOD

No.	SEX & AGE.	APEX BEAT.	THRILL.	AORTIC.			
				1ST.	2ND.	SYST.	DIAST.
2	F. 23	$\frac{6}{\text{Axilla}}$	O	O	O	X	X
3	F. 21	$\frac{5}{1\frac{1}{2}\text{ Extl. N.L.}}$	O	?	O	X	X
4	F. 18	$\frac{5}{2\text{ Extl. N.L.}}$	O	O	O	X	X
5	F. 20	$\frac{5}{\text{Just Extl. N.L.}}$	O	O	O	X	X
6	F. 50	$\frac{5}{1\frac{1}{4}\text{ Extl. N.L.}}$	O	O	O	X	X
7	M. 40	$\frac{5}{1\text{ Extl. N.L.}}$	O	O	O	O	X
8	M. 38	$\frac{6}{1\text{ Extl. N.L.}}$	O	O	O	X	X

TABLE II.—DISEASE OF THE AORTIC VALVES, WITH A MITRAL

No.	SEX & AGE.	APEX BEAT.	APICAL THRILL.	AORTIC.			
				1ST.	2ND.	SYST.	DIAST.
9	M. 26	O	O	O	O	X	X
10	M. 17	$\frac{6}{2\text{ Extl. N.L.}}$	Systolic	O	O	X	X
11	F. 42	$\frac{7}{\text{Ant. axilla}}$	Systolic	O	O	X	X
12	M. 35	$\frac{6}{\text{Ant. axilla}}$	O	O	O	X	X
13	M. 43	$\frac{5}{\text{Nipple line}}$	O	O	O	X	X
14	F. 15	$\frac{5}{1\text{ Extl. N.L.}}$	O	O	O	X	X
15	F. 18	$\frac{6}{2\frac{1}{2}\text{ Extl. N.L.}}$	O	O	O	X	X
16	M. 45	$\frac{6}{2\text{ Extl. N.L.}}$	O	O	O	X	X
17	M. 38	$\frac{5}{1\text{ Extl. N.L.}}$	O	O	O	X	X
18	M. 44	$\frac{5}{1\text{ Extl. N.L.}}$	O	O	O	X	X
19	M. 50	$\frac{5}{1\text{ Extl. N.L.}}$	O	O	O	X	X
20	F. 30	$\frac{5}{1\text{ Extl. N.L.}}$	O	O	O	X	O

In the third column the figure above the line represents the intercostal space in which represents in inches the distance outside the nipple line. Thus $\frac{6}{2\text{ N.L.}}$ = apex beat in

In the column devoted to the pulse, *regurgit* means that the pulse showed the

Post-mortem Examinations. Case No. 9.—Aortic valves incompetent; mitral valve valves incompetent; mitral valve admitted five fingers; left ventricle much hypertrophied three fingers with ease; left ventricle hypertrophied and dilated. Case No. 18.—Aortic cone, 1·55 in.]; left ventricle dilated and hypertrophied.

Apical First Sound and no Mitral Systolic Murmur.

Mitral. 1st.	Mitral. 2nd.	Mitral. Syst.	Pulm. 2nd.	Pulse.	Veins of Neck.	Liver	Referred Pain and Superficial Tenderness.
X	X	0	X	80 reg. regurgit.	0	+	Sudden attacks of pain. Tenderness Left Cerv. 3 to Dorsal 5.
X	X	0	+	80 reg. regurgit.	0	0	Left shoulder and arm to elbow. Tenderness C_4 to D_6. Mainly Left.
X	X	0	X	80 reg. regurgit.	0	0	Anginal attacks, *vide* § 7, p. 202.
X	X	0	X	100 reg. regurgit.	0	0	Left shoulder and arm to elbow. Left D_2 mainly, *vide* p. 162.
X	X	0	X	84 reg. regurgit.	0	0	L. neck, shoulder and arm to elbow. Tenderness Left C_4 and D_2.
X	X	0	X	100 reg. regurgit.	0	0	Sudden attacks on exertion. Widespread tenderness L. side.
X	X	0	X	100 reg. regurgit.	0	0	Left arm to elbow.

Systolic Murmur, abolishing the Apical First Sound.

Mitral. 1st.	Mitral. 2nd.	Mitral. Systolic.	Pulm. 2nd.	Pulse.	Veins of Neck.	Liver.	Referred Pain and Superficial Tenderness.
0	?	Conducted to angle of scap.	X	100 reg. regurgit.	0	0	No pain. No tenderness.
0	Feeble	Conducted to angle of scap.	+	108 irreg. regurgit.	0	0	No pain. No tenderness.
0	Feeble	Conducted to angle of scap.	X	100 reg. regurgit.	Pulsation	+	No pain. No tenderness.
0	0	Heard at angle of scap.	X	96 reg. regurgit.	0	0	No pain. No tenderness.
0	0	Heard to mid. axilla	X	92 reg. regurgit.	0	0	No pain. No tenderness.
0	X	Heard all over the back	+	120 reg. regurgit.	0	0	No pain. No tenderness.
0	2	Heard all over the back	+	96 reg. regurgit.	0	0	No pain. No tenderness.
0	X	Conducted to angle of scap.	X	80 reg. regurgit.	0	0	No pain. No tenderness.
0	X	Conducted to posterior axilla	X	68 reg. regurgit.	0	0	No pain. No tenderness.
0	X	Conducted to angle of scap.	X	84 reg. regurgit.	0	0	No pain. No tenderness.
0	0	Conducted to angle of scap.	X	80 reg. regurgit.	0	0	No pain. No tenderness.
0	X	Heard all over the back	+	88 irreg. low tension	Pulsation	0	No pain. No tenderness.

which the maximum pulsation of the apex is seen and felt. The figure below the line
th space, 2 in. outside the nipple line.

ow tension collapsing character typical of marked aortic regurgitation.
dmitted five fingers; left ventricle hypertrophied and dilated. Case No. 10.—Aortic
trophied and dilated. Case No. 11.—Aortic valves incompetent; mitral valve admitted
alves incompetent; mitral valve admitted five fingers [diameter, measured with the

physical signs exists from the beginning will not suffer from referred pain.

(3) The pain, when present, is mainly over the upper part of the chest and back, on the left side and in the left arm. It is accompanied by superficial tenderness over the second, third, and fourth dorsal, and sometimes the third and fourth cervical segmental areas.

(4) Headache over the frontal region is liable to be present. This is accompanied by tenderness over the frontal mid-orbital and fronto-temporal segmental areas on the scalp.

§ 4. *Pain produced by Aneurisms of the Aorta.*

The pain produced by an aneurism of the aorta is usually said to be due to: (1) pressure upon the walls of the chest and the vertebra, or (2) pressure upon nerve cords or roots. Now there is no doubt that these factors play a part in the production of the pain, especially when the aneurism has reached a great size. But in the case of smaller aneurisms, such pressure on surrounding structures plays a comparatively small part in the production of the pain. And yet pain is comparatively more common with the smaller aneurisms than with those of great size.

Now a careful examination shows that the pain is accompanied by superficial tenderness in a considerable proportion of cases of aneurism of moderate size. Moreover, this tenderness marks out areas on the surface of the chest and arm, which are to a great extent the same as those I have described above as concomitant phenomena of the pain of aortic disease. Thus this pain, accompanied as it is by superficial tenderness, is a simple referred visceral pain. For there can be no question of pressure on bones or nerves when the same pain arises as a sequel to aortic disease. And yet both aneurisms and aortic disease may cause pain accompanied by superficial tenderness over almost similar areas. We cannot wonder at this when we remember that etiologically the first part of the aorta is as much part of the heart as the ventricle.

The pain produced by aneurisms of the aorta must therefore always be considered under the following three groups: —

(1) True referred pain accompanied by superficial tenderness of the chest, neck, and arm.

(2) Referred sensations due to pressure on nerve trunks.

(3) Local pain produced by pressure on such structures as the vertebræ and ribs.

The commonest situation for the pain is down the inner side of the arm. Sometimes it only extends to the inner condyle of the humerus, but occasionally and not infrequently it extends over the ulnar side of the forearm to the little finger.

It is more common in the left arm than in the right. I cannot help thinking that if the pulsation of the aneurism appears to the right of the sternum, the pain will radiate down the right arm, whilst if it is visible to the left of the sternum, the left arm will be affected. I have not, however, at present sufficient material to make certain of this statement.

The pain is usually of a dull aching character, relieved by pressure or by rubbing. Thus patients who suffer from this pain frequently make the following characteristic movements: The left arm is somewhat adducted so that the elbow lies over the cardiac area of the chest. The right palm is placed over the left arm, so that the fingers reach over to that aspect of the left arm which is now outwards.[1] The right hand is then rubbed up and down over the outer part of the left arm.

Occasionally the pain is of a shooting character, and in that case it usually is said to shoot up from the elbow to the chest and back. If it extends to the little finger it shoots up from that finger to the elbow.

The pain sometimes assumes a paroxysmal character, and secondary anginal attacks may be developed. These are considered in § 7.

The pain is accompanied or followed by more or less marked superficial tenderness over the inner side of the arm, and sometimes over the ulnar side of the forearm to the little finger. When carefully marked out these areas

[1] Owing to the rotation of the left arm which accompanies the adduction this portion which now lies outermost is in reality the inner aspect of the arm.

correspond either to the maxima or to the full extent of the second and first dorsal segmental areas.

Now I pointed out in my first paper (p. 33) that the second dorsal area had a limb on the anterior surface of the chest, over the second space and third rib, and a dorsal limb, characterised by a spot close to the vertebral border of the spine of the scapula. In the same way the first dorsal area has a small portion on the back, at the level of the seventh cervical and first dorsal spine. Thus, if the whole of both these areas are represented, pain and tenderness will be present in the second space in front, and over the first and second dorsal spines, and the inter-vertebral groove to the left or the right of them.

Now in many cases this pain has been mistaken for a local pain, and has been ascribed to erosion of the chest in front, and of the vertebræ behind. But examination of the chest shows no signs of an aneurism of sufficient size to cause erosion of both the anterior and posterior walls of the chest. Moreover, the presence of the superficial tenderness shows that these spots are nothing more than the anterior and posterior limbs of segmental areas, of which the lateral limbs are to be found on the inner side of the arm.

But, it might be objected, that this pain down the inner side of the arm is of itself nothing but the expression of pressure on nerve trunks. But if this were so, it is difficult to see why it should appear in diseases of the aortic valves, following acute rheumatism (Case No. 5, p. 162). Again, pressure upon nerve trunks produces numbness and tingling, but not superficial tenderness, as anyone can learn who will compress his ulnar nerve and observe the result.

Moreover, the distribution of the hyperæsthesia is not that of any peripheral nerve or cord of the brachial plexus. Such a case as No. 21 (p. 171) is entirely incompatible with the supposition that this pain down the arm, to which I now refer, could be due to pressure upon nerve roots.

A most interesting motor phenomenon is occasionally associated with pain and superficial tenderness of the areas on the inner aspect of the arm and forearm. The patient complains that the hand feels stiff and numb. The grasp of

the hand is undoubtedly weaker when the pain is present than when it is absent. Thus I have seen a patient, in whom this pain came on owing to the effort of sitting up to eat his dinner, drop his fork from his left hand. When carefully tested, the movements of the fingers of the left hand were clumsy. There was no absolute paralysis, and no profound paresis that could be localised in any particular muscle, but yet all the fine movements of the left hand were badly performed. Movements of the thumb do not seem to

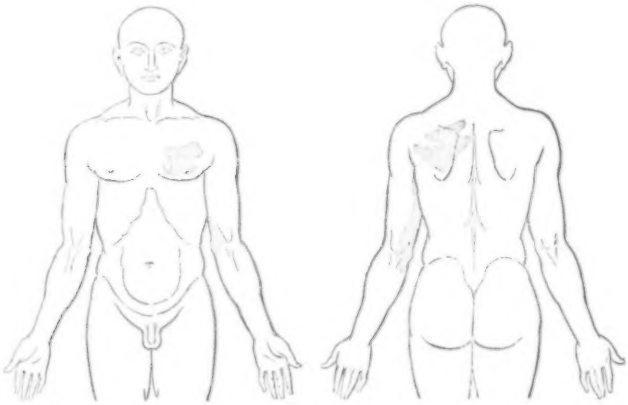


FIG. 2.

To show the superficial tenderness present in a Case of Aneurism of the Ascending Portion of the Aorta [Case No. 21].
The areas mainly affected belong to the 2nd and 3rd dorsal segments.

suffer to the same extent as fine movements of the fingers. This is a very transitory phenomenon, and soon disappears when the pain has gone. The interest of this motor condition lies in the fact that the first dorsal root with the eighth cervical supplies the small muscles of the hand. Thus an extreme disturbance on the sensory side, within the first dorsal segment, seems to cause some motor weakness within the area supplied by the first dorsal and eighth cervical root.

Case 21.—*To illustrate referred pain produced by aneurism of the ascending aorta.*—Charles T. (V.P.H., Dr. Eustace Smith). Aged 31, carpenter. Admitted, December 8, 1893.

An extremely strong healthy man till seven years ago, when he had a "hard chancre." Was treated with mercury. No secondary rash or other sequelæ.

Two years ago began to suffer from pain in the chest. He is a carpenter, and the pain came on particularly after using the heavy plane. September 22.—The pain became so bad he gave up work.

On admission.—He is a very finely built man of six feet. Face of a uniform pinkish colour, with no flush. No anæmia, jaundice, or œdema. Well nourished.

He is conscious of the pulsation of the swelling. No palpitation or fainting.

He states that three weeks before admission he suffered from violent pain in the left arm, which began in the second phalanx of the little finger and travelled upwards to the inner side of the elbow. The pain then extended as an intense aching over the whole of the ulnar half of the left forearm and in the little finger. Occasionally it darted and shot, and sometimes darted from the inner aspect of the elbow to the joint between the first and second phalanges of the little finger. This pain disappeared a week before admission, and has given place to the pain from which he now suffers. Pain is now present over the inner side of the arm as low as the fold of the elbow. Its point of greatest intensity is situated one inch (2.5 cm.) above the inner condyle of the humerus.

Some distinct superficial tenderness is present over an area extending upwards from the internal condyle of the humerus over the inner aspect of the left arm.

Visible pulsation in the first and second intercostal spaces. In the second space not only pulsation but a distinct systolic thrill can be felt.

The apex beat can be felt in the fifth space in the left nipple line. Impulse is feeble.

There is no bulging of the chest wall over the left front, but the percussion note is high pitched, and there is increased resistance over the upper part of the chest on the left side, extending from the first intercostal space to the fourth rib, and laterally from the left sternal border almost to the anterior axillary fold.

The cardiac dulness extends from the nipple line on the left to the mid-sternum on the right. Above, it merges into the dulness over the aneurism.

A loud systolic murmur is heard over the upper part of the chest on the left side, with its maximum in the second intercostal space. It is followed by an accentuated second sound.

Over the apex the first sound is weak, and is followed by a very soft systolic murmur, which disappears in the left axilla, but increases in intensity along the left border of the sternum to reach its maximum over the area of pulsation. Thus this murmur at the apex seems to be the conducted aneurismal murmur.

No diastolic murmur heard anywhere.

Pulse 80, regular. Artery large and full between the beats. The pulses seem to be equal.

No dyspnœa. No cough. Voice good. Larynx normal.

No abnormal signs in the lungs.

Tongue clean, appetite good. Bowels opened daily.

Liver not enlarged.

Urine 1020, acid. No albumen.

Pupils equal; react to light and accommodation. Both pupils dilate to shade.

December 13.—The pain extended down the ulnar side of the forearm to the little finger for a few hours.

December 14.—It is now represented by pain over the inner condyle, with definite superficial tenderness of inner aspect of arm.

The pulsation is decidedly more marked.

He was put strictly to bed, and the pulsation distinctly diminished, so that by March 5, 1894, the pulsating area in the second space could be covered by the top of the thumb.

Pain was practically absent throughout this period.

March 7.—He again began to complain of pain down the inner aspect of the arm, and marked superficial tenderness was present over the second dorsal segmental area. The pulsation is now visible over an area of three inches by one and a half inches (7·5 cm. by 3·75 cm.).

March 31.—The pulsating area has again increased, and has returned to the size on admission. The pulsation is accompanied by a marked systolic thrill, and the closure of the aorta semilunar valves can be felt over the pulsating area as a sharp slap. Apex beat and cardiac dulness as before.

Over the whole left side of the chest, as low as the fifth rib, a greatly accentuated second sound is audible. The systolic murmur is more widely heard over the upper part of the chest. No murmur or second sound audible in the back. Over the apex beat the first and second sounds are good, and are not obscured by the systolic murmur.

He now complains of pain—(1) over a spot just internal to the nipple in the third space. He places a single finger over this spot, and says that when the pain gets bad it enlarges. This pain passes through to a point in the back in the interscapular space, at a level of about the fourth dorsal spine. He says it is exactly as if someone were cutting him with a knife.

He also has some pain over the second rib, extending through to a point behind, close to the vertebral border of the spine of the scapula.

(2) He also has a dull aching "numb" feeling over the inner aspect of the left arm and ulnar aspect of the left forearm, to the ulnar side of the wrist, ulnar border of the hand, and to the head of the basal phalanx of the little finger. He is exceedingly precise in his description of the limits of this pain. When this

pain is bad he has loss of power in the fingers of the left hand, and very slightly in the thumb. The fingers feel "silly and useless." He could not grip his fork at dinner.

There is absolutely no loss of sensation to touch, pain, heat, or cold, but there is marked and definite superficial tenderness (hyper-algesia) over the areas in fig. 2.

Pulses equal. Pupils dilate to shade, and are equal. Larynx normal.

But with aneurism of the aorta, pain makes its appearance over other regions than these. Pain is sometimes situated in the left shoulder joint and in the supra-clavicular fossa and back of the neck. This pain may be accompanied by superficial tenderness over the third and fourth cervical areas as in the following case (No. 22). Now it is quite impossible that this pain above the clavicle could be due to pressure on nerve cords or roots, and yet, in this case, we see the pain and tenderness over the second and third dorsal merging imperceptibly into that above the clavicle.

Case 22.—*To illustrate the pain and tenderness over the cervical areas produced by aneurism of the aorta.*—James C. (V.P.H., Dr. Thorowgood), aged 43, sawdust dealer.

I first saw him December 11, 1893. He had suffered from pain in the back for six months. Six weeks before admission a cough came on, and the pain became much worse. He denies gonorrhœa and syphilis. Married, but wife has never been pregnant. No general illness. Not intemperate (two to three pints of beer daily).

On admission he complained of pain above and outside the left nipple, darting through to a point just below the spine of the scapula behind. He also had pain down the inner side of his arm to the elbow. Beautifully marked superficial tenderness was present over these spots.

The physical signs were those of aneurism of the aorta (see Part II. of this work, BRAIN, 1894, p. 371).

The right pulse was stronger than the left.

The pupils were normal and equal. Larynx unaffected.

He left the hospital free from pain, but on returning to work the pain and tenderness returned. But now the tenderness had spread to the fourth cervical area in the supra-clavicular fossa (*vide* fig. 16, p. 371, BRAIN, 1894).

Since this time he has frequently attempted to return to his somewhat heavy work, and each time the pain returned.

March 7, 1896.—The pain is distinctly increasing somewhat. It is still confined entirely to the left side of the chest, shooting through from the scapula to the upper spaces in front, from the

back of the neck to the left shoulder, and down the inner side of the arm to the elbow.

There is beautifully marked superficial tenderness as in fig. 3.

He complains of headache over the left half of the forehead, and this headache is accompanied by scalp tenderness, as in fig. 3.

No fainting. Occasionally feels faint and has to sit down. No marked palpitation. Becomes very short of breath on exertion.

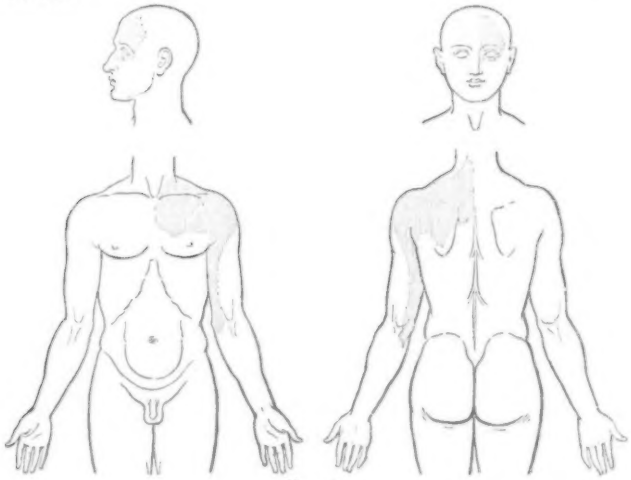


FIG. 3.

To show the superficial tenderness present in a Case of Aneurism of the Aorta [Case No. 22].

The areas mainly affected are those of the 4th cervical and 2nd and 3rd dorsal segments.

Tenderness of the scalp was present on the left side only, over the frontal, mid-orbital, and fronto-temporal areas.

Pulse 64; left pulse decidedly weaker than the right, all up the arm. No pulsation in the neck.

Cardiac pulsation in fifth space and left nipple line ($4\frac{3}{4}$ in. from middle line of sternum). Not heaving. No other pulsation visible or palpable over the chest.

Slight dulness or diminution of resonance over the upper part of the sternum. Cardiac dulness extends from left nipple line to right edge of sternum.

Over the upper part of the sternum and in the second and third right interspaces, a loud accentuated second sound is audible. This second sound is doubtfully heard to the left of the

second dorsal spine behind. A loud systolic murmur replaces the first sound over the aortic area. It is conducted down the left edge of the sternum. At the apex the first and second sounds are good. No murmur heard in the left axilla or at the angle of the left scapula. No tracheal tugging. Larynx normal. Pupils normal.

The same dulness at the right base to the angle of the right scapula, with absent breath-sounds and vocal resonance. These signs have remained unaltered since December, 1893.

This brings me to a point of much theoretical interest and importance. Some cases of aneurism, I believe mainly those which involve the arch of the aorta, complain of pain in the throat. This pain is described as "a kind of cramped feeling," "a sort of painful stiffness," and is situated in the space between the anterior border of the sterno-mastoid and the middle line on one or both sides. This pain is accompanied by superficial tenderness over an area which I have called the inferior laryngeal, an area which is shared by the structures of the larynx proper (fig. 4). Case No. 23 shewed this pain and superficial tenderness to a marked degree. I believe it is particularly liable to occur in those cases in which either the cervical sympathetic (shown by the pupil) or the recurrent laryngeal, or both nerves are affected, that is to say, where there are signs pointing to the arch of the aorta as the situation of the aneurism.

Case No. 23.—*To illustrate pain and tenderness over the areas of the front of the neck in aneurism of the aortic arch.*—Jane R. (Marylebone Infirmary, Mr. Lunn), aged 53 years, married, charwoman. Admitted November 17, 1895.

October, 1894.—She had a cough and some pain in the back. This did not disappear, and in January, 1895, she went to the Brompton Hospital and was admitted. Thence she was transferred to the Middlesex Hospital, where she remained till her admission to the Infirmary. Throughout this time she has suffered from more or less pain. When she got up the pain grew worse; when she lay in bed it was easier.

Menstruation ceased twelve years ago. Married at 21. Never pregnant. No miscarriages. Never any discharge from the vagina.

Temperate. No history of acute rheumatism or any illness except, possibly, typhoid fever.

On Admission.—She is a well-built, bright, cheerful looking woman of 52. Looks somewhat older. No anæmia. No jaundice. No œdema. Is not wasted.

She says that her heart beats very fast on exertion, but never seems to stop or beat irregularly. This rapid beating is not accompanied by any pain. No shortness of breath whilst in bed. She can lie flat at night.

She has no pain now, and there are no signs of any definite superficial tenderness. No headache.

Pulse 68, regular. Rather small stroke but of well maintained tension. The arterial wall is a little hard. The right pulse is if anything a little stronger than the left.

Apex beat cannot be definitely localised. No thrill. No obvious pulsation seen or felt over the upper part of the chest.

Cardiac dulness begins in the third space, extends $3\frac{1}{4}$ in. (8 cm.) to the left (left nipple line), and to the middle of the sternum on the right. No dulness elsewhere over the chest.

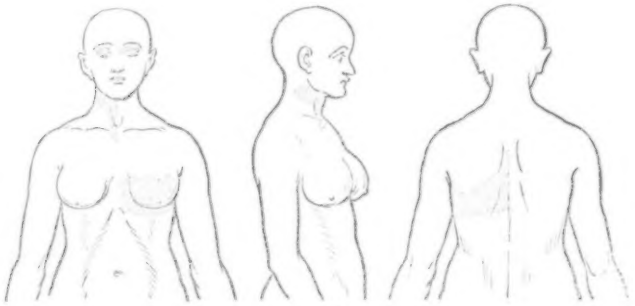


FIG. 4.

To illustrate the pain and tenderness over the Inferior Laryngeal Area of the Neck sometimes present in Aneurism of the Arch of the Aorta [Case No. 23].

Superficial tenderness was also present in this case over the 5th and 6th dorsal areas on the left side of the chest and back.

Over the upper part of, and especially to the right of the sternum, an accentuated slapping second sound is heard. Over almost the same area a soft blurring systolic murmur is audible which is conducted slightly towards the apex. The first sound over the aortic area is feeble. Over the apex the systolic murmur is just audible, and the first and second sounds are good. Neither the second sound nor the systolic murmur are heard anywhere in the back.

No cough now. No marked abnormality in the physical signs of the lungs.

Liver not enlarged.

The voice is a little hoarse, no tracheal tugging. The left vocal cord does not move so well on phonation as the right.

The left pupil under ordinary circumstances is almost 3 mm. in diameter, and is equal to the right. It practically does not react to light or dilate to shade. With a strong light the difference between the pupils becomes very marked. The left pupil dilates somewhat, but not to the extent of the right, under the influence of cocaine. Both pupils react well to accommodation. There is no marked difference in the size of the two palpebral fissures or in the projection of the eyes.

Urine normal.

She remained absolutely in the above condition till the afternoon of January 14, 1896. She then suddenly began to scream with the pain in the head and abdomen. She complained of pain across the upper part of the abdomen on the left side, and over the back below the left shoulder blade. Intense widespread superficial tenderness of chest and back, more marked on the left side. Very marked tenderness over the frontal, mid-orbital, fronto-temporal and temporal areas of the scalp.

Pulse 64, with the same doubtful difference between the two sides.

There is now a distinct fulness over that part of the first and second intercostal spaces on the right side, which lies on each side of the second rib cartilage; thus the second rib cartilage has the appearance of being bulged. A systolic impulse and at times a systolic thrill is felt over this area, followed by a shock corresponding to the second sound.

A loud systolic murmur is heard over the second right rib cartilage, followed by a loud bell-like second sound.

The cardiac signs as before. No sign of dilatation of the left ventricle.

Nothing heard elsewhere in the chest.

The left pupil is markedly smaller than the right, and does not dilate at all to shade.

From this point onwards the pain and superficial tenderness lay mainly within the fifth, sixth, and seventh dorsal areas on the left side. (Fig. 4.) The headache and scalp tenderness were more temporal than before.

March 11.—She still has the pain and tenderness in the left side, but she has just begun to complain of a fresh pain on the right side of the throat. She places her right hand flat over the right side of the space between the anterior borders of the sterno-mastoids, the fingers almost touching the lobule of her ear, and the ulnar border of the hand parallel to the lower border of the jaw. She complains that it is an aching pain like the pain on the left side of the back.

There is more exquisitely marked superficial tenderness over the right inferior laryngeal area (*vide* fig. 4). On the left side of the trunk is seen the tenderness which has been present ever since the attack on January 14.

Occasional slight cough. No alteration in the voice. No marked difference in the movements of the two vocal cords. No

intra-laryngeal cause for the pain discoverable. Cords a normal colour.

Pulse of small stroke. The right pulse is slightly stronger than the left.

There is slight but distinct dulness in the first and second spaces on the right side. Over this area a distinct systolic thrill can be felt. This dulness joins with the upper limit of cardiac dulness across the sternum.

The same systolic murmur and markedly accentuated second sound are audible over the upper part of the right chest.

The left pupil acts to accommodation, but does not dilate in the least to shade.

She became freer of pain, but slight pain and superficial tenderness were still present over the right infra-laryngeal area on April 13, 1896.

The pain of aneurism usually improves greatly under rest and treatment, and all pain and tenderness may disappear after a time. But these cases are notoriously disappointing. Physician and patient are full of hope, when, without obvious cause, or perhaps in consequence of some increased exertion, the pain suddenly returns, not uncommonly with great violence. Suppose the pain and tenderness lay originally over the upper areas of the chest and down the inner side of the arm; with a sudden return of this kind, the pain and tenderness may no longer lie over the upper part of the chest, but may occupy areas under the heart, in the epigastrium and below the shoulder blade (case 23). The tenderness, which previously lay within the fourth cervical and first and second dorsal segments, may now occupy the fifth, sixth or seventh dorsal.

Now we cannot suppose that a new aneurism has suddenly sprung up four inches lower down in the region of the diaphragm, which is above the level of the new outburst of pain. Moreover, the superficial tenderness shows that the pain is referred and not local. Therefore I think we must assume that that portion of the aorta which causes referred pain over the first and second dorsal segments and over the inferior laryngeal area, lies close to that part which causes referred pain over the fifth, sixth and seventh dorsal segments. Now this distribution of pain and tenderness seems to me peculiarly liable to be present in those aneurisms which involve the cervical sympathetic or the recurrent laryngeal,

that is to say, aneurisms of the arch. I shall show reason to believe that the aorta beyond the position of the ductus arteriosus refers into the mid-dorsal region, whilst the aortic bulb and arch refer on to the higher dorsal and inferior laryngeal areas.

Some aneurisms are painless from the beginning or become so in course of time, and many observers have commented on the absence of pain in aneurisms sufficiently large to cause marked symptoms of pressure on the trachea and bronchi. I give as an instance of such a condition case No. 24, where, though there was a history of pain, no referred pain or tenderness was present during the ten weeks he was under observation.

The explanation of this absence of referred pain and superficial tenderness seems to me to be as follows:—The walls of such aneurisms are composed of matted connective tissue, and all true aortic wall has long ago disappeared. The aorta is markedly atheromatous and it is these atheromatous patches which yield to the pressure within. Connective tissue becomes thickened around the yielding patch on the outer side of the aortic wall, and thus the aneurism gradually grows, not by a true distension of the aortic wall, but by the stretching of a wall composed of connective tissue. Thus the wall of the organ, the integrity of which is necessary for the production of referred pain, is no longer present, or is only present in such a condition that the nerves are long past transmitting afferent impulses to the central sympathetic system. Thus such an aneurism may cause local pain by pressure on the structures within the chest, or pain and anæsthesia in the course of nerve cords or roots, but will not cause referred visceral pain accompanied by superficial tenderness.

Case No. 24.—*To show how an aneurism that causes marked pressure symptoms may be painless.*—William M. (V. P. H. Dr. Eustace Smith) age 53, an engine fitter.

In 1892 he began to suffer from constant pain in the right axilla. This gradually died away. In February, 1893, after all pain had died away, he began to suffer from shortness of breath which gradually became constant. He then began to cough, and

during the summer of 1893 noticed that his expectoration was occasionally streaked with blood.

At the age of 23 he says he had syphilis. No secondary symptoms. Has drunk to excess occasionally. No gout. No rheumatism.

On Admission (*February* 7, 1894).—Medium-sized man of 53. Face pale and blotchy, with dilated vessels on the cheeks but no true flush. Lips and ears a little blue. Fingers and toes much clubbed. Lower part of the back œdematous.

He complains of palpitation, but only on exertion. It did not cause pain. Short of breath even when in bed; he cannot sleep except when propped up.

He complains of a pain on exertion situated over the epigastrium. He has no other pain. No pain in the arm or upper part of the chest. There is no superficial tenderness anywhere, not even over the situation of the epigastric pain. No headache. No scalp tenderness.

Pulse 88, regular, fair stroke and good tension. Arterial wall distinctly hard. Pulses equal. No capillary pulsation. No venous pulsation in the neck.

General heaving impulse in fourth and fifth spaces. No definite apex beat. No pulsation visible elsewhere. No thrill.

Cardiac dulness much diminished. No dulness over the upper part of the chest.

Over the upper part of the sternum and to either side of it is heard a low-pitched systolic murmur. This murmur is conducted down the sternum, but is not heard at the apex of the heart. It is heard in the left interscapular fossa and over the spine with greater intensity than in front. The aortic second sound is heard but is not increased. Pulmonary second sound short and sharp. At the apex first sound is booming, second sound good.

Respiration about 30. Distinct dyspnœa. Alæ nasi dilating and sterno-mastoids acting. Harsh, dry, tearing cough, more troublesome at night. He brings up a small quantity of tough, aerated mucous expectoration.

Chest somewhat rigid. Movements very poor at both bases, but about equal on the two sides. Hyper-resonant note all over the chest. Breath sounds and vocal resonance good at apices but weak over both bases. Moist râles over both bases.

Tongue red, fairly clean and moist, no pain after food now. Bowels confined.

Liver can just be felt. Spleen not felt.

Urine, sp. gr. 1025, acid. No albumen. No sugar.

He had a very irregular temperature, rising sometimes to 102° F. (39° C.), but frequently not reaching 100 as the highest point in the day. He began to spit up small round masses of muco-purulent material in considerable quantity, and signs first of consolidation and then of excavation began to appear over the right lung. By April 15 the whole right side was implicated, and he died on April 18.

Throughout the whole of this time he suffered from no pain, except that at the epigastrium. No referred headache or tenderness.

Post-mortem.—A sacculated aneurism was found about the size of a large orange. It began below, about $\frac{3}{4}$ in. (2 cm.) above the aortic valves, and involved the greater part of the transverse arch of the aorta, including the origin of the innominate and left carotid arteries. The right branch of the pulmonary artery was extremely flattened and seemed to be running in the substance which formed the wall of the aneurism. Its lumen was almost completely occluded. The left branch of the pulmonary artery was stretched over the front of the aneurism, and its lumen was diminished but not occluded. The aneurism was filled with laminated clot and its walls showed no recognisable remains of the normal aortic lining. They were formed of tough more or less uniform connective tissue. Where not affected by the aneurism the aorta was intensely degenerated. The wall contains much calcareous matter, and in places the coats are infiltrated with blood. Right ventricle much dilated and hypertrophied. Left ventricle small, normal. Aortic valves slightly atheromatous but competent. Mitral valves normal.

Right lung contained one large ragged walled cavity filled with blood-stained stinking grumous fluid. The greater part of the lung was gangrenous, apparently from occlusion of the right pulmonary artery. Left lung normal.

To sum up the conclusions to which we have arrived in this section:—

(1) Aneurism of the aorta may cause referred pain, associated with superficial tenderness over the following areas:—(*a*) Third and fourth cervical and first, second and third, and occasionally fourth, dorsal segmental areas usually on the left side but occasionally on the right side. (*b*) Over the inferior laryngeal area on one or both sides. (*c*) Over the areas from the sixth to the eighth dorsal. These areas appear when we have reason to suppose the aneurism has spread beyond the point where the ductus arteriosus enters the aorta.

(2) Aneurism of the aorta may cause local pain from pressure on structures in the chest other than nerves.

(3) Aneurism of the aorta may cause pain, and occasionally, though rarely, anæsthesia from pressure on peripheral nerves or roots. Such pain is not associated with superficial tenderness over segmental areas. Contrary to the

usual opinion this is the least common cause of pain in aneurism.

(4) Aneurisms which arise in a highly diseased aorta (case 24) or such as are bounded by matted connective tissue only, cause only local and no true referred visceral pain.

§ 5. *Referred Pain in Disease of the Mitral Valves.*

There are no lesions about which greater difference of opinion exists than those of the mitral valve. For at one time the presence of a systolic murmur over the apex in organic cardiac disease at once led to the diagnosis of mitral regurgitation. That the murmur is due to some regurgitation through the mitral orifice is highly probable, but we want to know whether it is the main lesion or not. Broadbent and Graham Steell[1] have long taught that this murmur alone tells us very little with regard to the condition of the mitral orifice, and that the important fact is the relation of the murmur to the first sound of the heart. Thus the only sign of marked mitral stenosis may be a systolic murmur accompanied by a sharp first sound at the apex. In such a case the murmur is that of regurgitation, but the important pathological factor in the physics of the circulation is stenosis of the mitral orifice.

In the course of this research I soon found that it was quite impossible to classify my observations on the referred pain that occurred with lesions of the mitral valves according to the name of the supposed valvular lesion. For in any one case competent observers would disagree in how far regurgitation or stenosis were the main factor. Now the presence or absence of referred pain depends on the conditions within the cavities of the heart. As these conditions can only be inferred from a knowledge of the conditions of the valves, such fundamental differences of opinion amongst experts formed a severe hindrance to me in the earlier stages of this work. With regard, however, to the actual physical signs, I found no such difference of opinion; the difference lay rather in the interpretation of these signs.

[1] Broadbent, *American Journal of Med. Sciences*, No. 181, n.s., Jan., 1886. Graham Steell, *Med. Chronicle*, May, 1888; Sept., 1895.

Throughout this section I shall therefore speak as little as possible of mitral stenosis or mitral regurgitation, but shall attempt to show that, with lesions of the mitral valve, the presence of certain conjunctions of physical signs is associated with referred pain, whilst when certain other physical signs are present, pain is absent. My ideas of the significance of these physical signs are relegated to a special theoretical chapter (chap. ii.).

By adopting this plan of speaking in terms of physical signs I hope that my observations will stand the test of time, though my ideas of the significance of these signs may be falsified by future research. I am certain that if this plan had been adopted by all writers on the subject of mitral disease, much of the present confusion would have been avoided.

In this section I shall only treat of those cases in which the right heart has not failed. For, as soon as the liver begins to be enlarged, pain may make its appearance which is not directly due to the heart. Such cases are reserved for § 6. Thus the auscultatory signs which most concern us here are the mitral diastolic, præsystolic, and systolic murmurs, and the conditions of the apical first and second sounds. Of the diastolic and præsystolic murmurs nothing need be said, but a few words are necessary about the systolic apex murmur.

A systolic murmur heard at the apex may weaken or abolish the first sound, so that the first sound may be absent over the apex, in the axilla, and at the angle of the left scapula. On the other hand, the same murmur may be accompanied by a short, sharp, first sound, such as is usually heard in conjunction with a præsystolic mitral murmur. As Steell[1] has pointed out, a systolic murmur of this kind, associated with a sharp, first sound, is usually not conducted into the axilla, or heard at the angle of the left scapula. But he recognises that this conduction or non-conduction to the angle of the left scapula is an unsafe guide. Thus the real point of importance is not the conduction of the murmur, but the fact that it is associated with a sharp

[1] *Med. Chronicle*, May, 1888, and Sept., 1895.

first sound, either at the apex or in the axilla. That these observations of Broadbent and Steell are of vital physiological importance, and not simply of academic interest, is borne out by the value of these signs as a basis for the classification of a physiological condition such as referred pain.

(*a*) Whenever the signs of old endocardial mischief consist solely of a systolic murmur heard at the apex, and no first sound is heard either at the apex or in the axilla, or at the angle of the left scapular, referred pain is absent as a marked feature of the case.

I carefully exclude from this statement all cases where a systolic murmur is heard owing to the failure of a heart that has hypertrophied from other causes than endocarditis (*e.g.*, chronic Bright's disease). I also exclude those cases of acute rheumatism in which a systolic murmur appears during the attack. Table III., p. 186, shows a series of cases in which these conditions were fulfilled. They exhibited neither referred pain, referred headache, nor superficial tenderness of the chest or scalp throughout a period of observation extending from a minimum of six weeks up to a year.

Such cases, however, suffer from palpitation on exertion, and this palpitation is accompanied by shortness of breath and occasionally by a pain which is said to be situated over the apex of the heart. This pain does not go through to the back or round the body like a referred pain, and is unaccompanied by headache or by superficial tenderness of the trunk or scalp. It is apparently a true local pain.

(*b*) The second group of cases consists of those in which a mitral diastolic murmur is audible together with a systolic murmur. The systolic murmur is widely heard and conducted to the angle of the left scapula. No first sound is audible either at the apex or in the axilla, or at the angle of the left scapula.

Such physical signs are not associated with referred pain connected with the heart. As, however, the later stages of mitral stenosis fall into this group, referred pain may be

TABLE III.—MITRAL SYSTOLIC MURMURS AT THE APEX, OBSCURING THE

No.	Sex & Age.	Apex Beat.	Apical Thrill.	APEX.			
				1st.	2nd.	Systolic.	Diast
25	F. 17	$\frac{5}{\text{Just Extl. N.L.}}$	Systolic	O	O	Heard widely over back	O
26	F. 14	$\frac{5}{\text{1 Extl. N.L.}}$	O	O	X	Conducted to angle of scap.	O
27	F. 12	$\frac{5}{\frac{1}{2}\text{ Extl. N.L.}}$	Systolic	O	X	Heard widely over back	O
28	F. 21	$\frac{5}{\text{In N.L.}}$	O	O	X	Heard widely over back	O
29	F. 23	$\frac{5}{\frac{1}{2}\text{ Extl. N.L.}}$	O	O	2	Heard widely over back	O
30	M. 16	$\frac{5}{\text{In N.L.}}$	Systolic	O	2	Conducted to angle of scap.	O

TABLE IV.—MITRAL DIASTOLIC ACCOMPANIED BY SYSTOLIC MURMUR AT THE A

No.	Sex & Age.	Apex Beat.	Apical Thrill.	APEX.			
				1st.	2nd.	Systolic.	Diast.
31	M. 23	$\frac{5}{\text{In N.L.}}$	Diastolic	O	2	Conducted to angle of scap.	X
32	F. 19	$\frac{5}{\text{In N.L.}}$	O	O	X	Conducted to angle of scap.	X
33	M. 42	$\frac{6}{\text{1 Extl. N.L.}}$	O	O	2	Conducted to angle of scap.	X
34	M. 18	$\frac{5}{\text{1 Extl. N.L.}}$	Diastolic	O	2	Conducted to angle of scap.	X
35	F. 17	$\frac{6}{\text{1 Extl. N.L.}}$	Systolic	O	O	Conducted to angle of scap.	X
36	F. 18	$\frac{6}{\frac{1}{2}\text{ Extl. N.L.}}$	O	O	O	Conducted to angle of scap.	X

The abbreviations are the same as on Tables I. and II.

In the column devoted to the Pulse *Maintained*, means that although the pulse-stroke is "Virtual Tension"). On the other hand, *low tension* means that the pulse is compressible.

In the column devoted to the 2nd sound, 2 means that this sound is re-duplicated.

In the column devoted to the 1st sound at the apex, + means that this sound

THE FIRST SOUND AT THE APEX, AND IN THE LEFT AXILLA.

Diast.	Presystolic.	Pulm. 2nd Sound	Pulse.	Veins of Neck.	Liver	Lungs.	Referred Pain and Tenderness.
0	0	+	100 reg. low tension	0	0	Crackles at bases	No pain. No tenderness.
0	0	+	84 reg. low tension	Slight pulsation	0	R. base crackles	No pain. No tenderness.
0	0	+	120 reg. low tension	Slight pulsation	0	Crackles at bases	No pain. No tenderness.
0	0	+	116 reg. low tension	Slight pulsation	0	0	No pain. No tenderness.
0	0	-	84 reg. low tension	0	0	0	No pain. No tenderness.
0	0	2+	66 reg. low tension	Slight pulsation	0	0	No pain. No tenderness.

THE APEX, OBSCURING THE FIRST SOUND AT THE APEX AND IN THE AXILLA.

Diast.	Presystolic.	Pulm. 2nd Sound	Pulse.	Veins of Neck.	Liver	Lungs.	Referred Pain and Tenderness.
X	0	+	74 irreg. maintained	0	0	0	No pain. No tenderness.
X	0	+	120 reg.	0	0	0	No pain. No tenderness.
X	0	+	60 irreg.	0	0	Crackles both bases	No pain. No tenderness.
X	0	+	88 reg. maintained	0	0	L. base	No pain. No tenderness.
X	0	+	76 reg.	0	0	Crackles L. base	No pain. No tenderness.
X	0	2	52 irreg.	0	0	0	No pain. No tenderness.

...stroke is small, the tension, such as it is, is maintained between the beats (Broadbent's ...ssible, and whatever the initial stroke the tension is low between the beats. ...- means that the 2nd sound is re-duplicated, but one element is accentuated. ...rt, sharp, and high-pitched, as is typically found in marked mitral stenosis.

TABLE V.—SYSTOLIC MURMURS HEARD AT THE APEX TOGE

No.	SEX & AGE.	APEX BEAT.	APICAL THRILL.	APEX.			
				1ST.	2ND.	SYSTOLIC.	DIAS
37	M. 21	$\frac{5}{\text{Intl. N.I.}}$	O	+	X	Not conducted	X
38	F. 36	$\frac{5}{\text{Extl. N.I.}}$	O	+	X	Not conducted	O
39	F. 22	$\frac{5}{\frac{1}{2}\text{ Extl. N.I.}}$	Presystolic	+	X	Conducted	O
40	F. 29	$\frac{5}{\text{Intl. N.I.}}$	Diastolic	+	X	Not conducted	X
41	F. 33	$\frac{5}{\text{Intl. N.I.}}$	O	+	X	Not conducted	O
42	F. 17	$\frac{5}{1\text{ Extl. N.I.}}$	Diastolic	+	2	Conducted	X
43	M. 42	$\frac{5}{\text{Intl. N.I.}}$	O	+	X	Not conducted	
44	F. 38	$\frac{5}{\text{Intl. N.I.}}$	Systolic	+	X	Not conducted	O
45	F. 42	$\frac{5}{\text{In N.I.}}$	Systolic	+	X	Not conducted	X
46	F. 36	$\frac{5}{1\text{ Extl. N.I.}}$	Diastolic	+	?	Not conducted	X

TABLE VI.—MITRAL DIASTOLIC OR PRESYSTOLIC MURMUR. MITRA

No.	SEX & AGE.	APEX BEAT.	APICAL THRILL.	APEX.			
				1ST.	2ND.	SYSTOLIC.	DIAST
47	F. 24	$-\frac{5}{\text{N.I.}}$	O	+	X	O	O
48	M. 27	$\frac{5}{\text{N.I.}}$	O	+	X	O	X
49	M. 30	$\frac{5}{\text{N.I.}}$	Diastolic	+	X	O	X
50	M. 30	$\frac{5}{\text{Intl. N.I.}}$	O	+	X	O	O
51	M. 37	$\frac{5}{\text{Intl. N.I.}}$	Presystolic	+	X	O	O
52	F. 49	$\frac{5}{\text{N.I.}}$	Presystolic	+	X	O	O
53	F. 50	$\frac{5}{\text{N.I.}}$	O	+	X	O	X

The abbreviations are the same as on Tables I. and II.

In the column devoted to the Pulse *Maintained*, means that although the pul- rake is "Virtual Tension"]. On the other hand, *low tension* means that the pulse is com- ssible

In the column devoted to the 2nd sound, 2 means that this sound is re-duplicated - mea

In the column devoted to the 1st sound at the apex, + means that this sound s ort, sh

Post Mortem Examinations. Case No. 43.—Mitral valve much stenosed [diamet easure Mitral valve much stenosed; converted into flattened funnel [diameter with cr 8 in.]

TOGETHER WITH A SHORT, SHARP, FIRST SOUND.

Diast.	Presys-tolic.	Pulm. 2nd Sound	Pulse.	Veins of Neck.	Liver	Lungs.	Referred Pain and Tenderness.
X	?	+	76 reg. maintained	O	O	O	Stomach, back, tmpls. L. D_6 & D_7
O	O	+	100 reg. maintained	Pulsation	O	O	Stomach & back. D_5 to D_8 L. & R.
O	X	+	100 reg. maintained	O	O	Crackles both bases	Stomach & back. L. D_6 and D_7.
X	X	+	64 reg. maintained	O	O	Crackles both bases	Stomach & back. D_6 and D_7 R. & L.
O	O	+	76 reg. maintained	O	O	O	L. breast & back. L. D_5 to D_8.
X	O	+	88 irreg. maintained	Pulsation	O	Crackles both bases	L. breast & back. L. D_6.
	O	+	88 irreg. maintained	O	O	Crackles both bases	Stomach & back. L. D_6 and D_7.
O	O	+	100 reg. maintained	O	O	Crackles both bases	L. breast & back. L. D_5, D_6, D_7.
X	O	+	76 reg. maintained	O	O	O	L. breast & back. L. D_6.
X	X	+	80 irreg. maintained	Slight pulsation	O	Crackles both bases	Stomach & back. L. D_5, D_6 D_7.

Mitral Systolic. First Sound Short and Sharp at Apex.

Diast.	Presys-tolic.	Pulm. 2nd Sound	Pulse.	Veins of Neck.	Liver	Lungs.	Referred Pain and Tenderness.
O	X	+	70 reg. maintained	O	O	O	On exertion under L. breast & back.
X	X	2+	60 irreg. maintained	O	O	O	On exertion under L. breast & back.
X	X	+	54 reg. maintained	O	O	O	On exertion only.
O	X	+	80 irreg. maintained	O	O	O	On exertion.
O	X	+	80 irreg. maintained	O	O	Crackles at bases	O
O	X	+	84 irreg. maintained	O	O	O	O
X	X	+	60 irreg. maintained	O	O	O	O

stroke is small, the tension, such as it is, is maintained between the beats [Broadbent's
possible, and whatever the initial stroke the tension is low between the beats.
– means that the 2nd sound is re-duplicated, but one element is accentuated.
short, sharp, and high-pitched, as is typically found in marked mitral stenosis.
measured with cone 0·8 in.]. Left ventricle somewhat hypertrophied. Case No. 51.—
·8 in.]. Left ventricle not hypertrophied.

present from enlargement of the liver, or affection of other organs than the heart. I have therefore not included such cases of cardiac failure in the short tabular arrangement (Table IV.) that I have drawn up to illustrate this statement.

(*c*) When a mitral systolic murmur is heard together with a sharp first sound at the apex or in the axilla, referred pain tends to appear as a symptom of the disease. Such a systolic murmur is usually not conducted into the axilla or heard at the angle of the left scapula, but this rule is frequently broken. It is, however, rarely if ever heard all over the back, and when heard at the angle of the left scapula is not infrequently accompanied by the first sound. This class of case may occasionally be characterised only by the above-mentioned signs, but usually a presystolic or diastolic murmur, and occasionally both murmurs are heard in addition to the systolic apical murmur. [Table V.]

By the statement that referred pain tends to appear in this class of case, I do not mean that it is continuously present. Thus, when the patient is at rest it is usually absent; but any undue exertion or any deterioration in the general condition will be signalised by the appearance of referred pain and headache. Moreover, a complaint of more or less pain is a feature in the history of such cases if they are watched over a considerable period of time.

(*d*.) A fourth group of cases of mitral disease consists of those in which no systolic mitral murmur is present. Either a presystolic, a diastolic, or both murmurs may be present, and the first sound has the typical short, sharp character usual in stenosis. The second sound is usually heard at the apex, and such cases thus belong to what Broadbent calls the first stage of mitral stenosis. [Table VI.]

In cases of this kind referred pain is not usually present. It may make its appearance as the sequel of some unusual act of exertion, or on first getting up after a considerable rest in bed; but nevertheless, referred pain is the exception rather than the rule during the time these signs are actually present.

I have so far spoken of referred pain in disease of the mitral valve without mentioning its position or characteristics. In such lesions as cause pain, it is situated within an area bounded above by a horizontal line at the level of the nipples and below by the line from the umbilicus to the costal border, and thence straight to the centre of the back. Not that the pain occupies the whole of this area at any time. It is mostly situated in the fifth space below the heart, at the epigastrium, and in the back just below or just internal to the angle of the scapula. It is situated mainly on the left side of the chest and back, sometimes entirely on the left side. The pain is not usually sharp and darting, but is, as a rule, dull and aching in character. It never becomes paroxysmal, or shows a tendency to spread suddenly and widely like the pain of aortic disease. It disables the patient less, but at the same time is more constant than the pain of aortic disease.

This pain is accompanied by superficial tenderness of greater or less extent over the sixth, seventh, and eighth dorsal segmental areas. The ninth dorsal also appears to be occasionally affected, but never without one or more of the above-mentioned areas. I therefore feel inclined to think that the implication of the ninth dorsal is due to spread downwards.

This tenderness is always worse on the left side, and the tenderness on the left side is the first to appear and the last to disappear.

The headache is situated in the temples and on the vertex, and more or less superficial tenderness is usually present over the temporal, vertical, parietal, segmental areas of the scalp.

Case 43, *to illustrate referred pain produced by lesions of the mitral valve.*—Peter H., aged 42. In 1882 he had rheumatic fever in which his feet and ankles were swollen and painful.

In 1892, one evening, after coming from work he spat a little blood. A month later he again spat a little blood after coming from work. He had suffered from pain in the chest for some time, and at last was compelled to go to a doctor (at the end of 1892), who said his heart was affected. He has been under the

doctor on and off ever since. He drank somewhat heavily till 1886, when he married; since then has been sober.

December 19, 1894.—Medium-sized man of 42; looks about his age. Face dusky, with a blue-red flush on both cheeks. No cyanosis of extremities, no œdema, no jaundice, no obvious dyspnœa. Says he becomes short of breath if he exerts himself much. Lies on his right side in bed. He complains of pain on

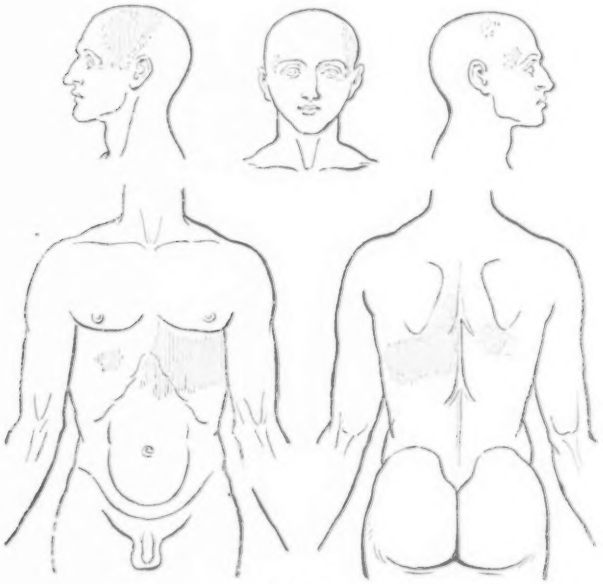


FIG. 5.

To show the extent of the superficial tenderness in a Case of Mitral Stenosis [Case No. 43], characterised by the following signs—a sharp first sound at the apex, followed by a systolic murmur not conducted to the angle of the left scapula.

The areas mainly affected on the trunk are the 6th and 7th dorsal on the left side. On the scalp the temporal and vertical areas are affected, mainly on the left side.

the left side of the chest in the fifth and sixth interspaces, and in the back close to the angle of the scapula. There is distinct superficial tenderness over both points.

Pulse 80, irregular both in rate and force. The irregularity in force is more marked than the irregularity in rhythm. Arterial wall not hard.

No pulsation of veins in the neck. Heart's apex beat seen and felt in fifth space 4½ inches from the middle line (11·5 cm.), just outside the left nipple line. Impulse weak and irregular; no thrill.

Cardiac dulness begins above at the third space, extends about 4½ inches (11·5 cm.) to the left and to the mid-sternum on the right.

Over the apex, the first sound is distinct, but sharp and thin; but further to the left in the axilla the first sound is distinctly sharp and high-pitched. The second sound is repudiated. A systolic murmur is audible over the fifth and sixth spaces from the left edge of the sternum to the line of the anterior axillary fold. Its maximum intensity lies over the apex beat, and no murmur is audible in the mid-axilla or at the angle of the left scapula. *Over the left second and third spaces* the second sound is accentuated; *over the right second and third spaces* the first sound is weak, the second sound somewhat slapping.

Respiration 24, regular. Alæ nasi and accessory muscles of inspiration are not working. No cough heard; no expectoration.

There are no abnormal signs in the lungs beyond those of slight general emphysema and a few moist crackling sounds at both bases.

Tongue clean, not furred. Appetite good, no vomiting; no pyrosis or eructation. Bowels opened daily. Urine 1020 acid. No albumen.

January 3.—He complained of pain in the epigastrium and below the angle of the left scapula. Marked band of superficial tenderness marking out the seventh dorsal area. Headache over the left temple with superficial tenderness over the left temporal area. Pulse 88, very irregular. Tongue clear, no vomiting. Physical signs as below.

January 4.—Exactly the same pains, headache and tenderness.

January 5.—The pain and headache are more marked, but are still practically confined to the left side.

The superficial tenderness both of the trunk and scarp are now very marked (fig. 5), and slight spots of tenderness have begun to appear on the right side. Tongue clean, moist. The pain is somewhat increased after taking food, but it is always there whether he takes food or not, and all food of every kind increases it. No vomiting; bowels still opened daily.

Pulse very irregular and of small stroke. No pulsation of veins in neck. Cardiac impulse felt diffusely in fifth space with its maximum above in his nipple line. Some epigastric pulsation. Suspicion of an occasional diastolic thirst.

Cardiac dulness beginning above in third space, extends about four inches to the left and about half-inch to the right of middle line of chest.

Over apex, first sound accentuated and sharp, second feeble. Systolic murmur not conducted into axilla or heard at angle of

left scapula. *Over left second and third spaces* second sound less markedly accentuated than before; *over right second and third spaces* first sound heard, second weak. Signs in lungs as before.

January 9.—The pain and tenderness became more accentuated, but occupied the same area as in fig. 5, except that the tenderness is more marked over the right side than in that figure.

January 15.—He continued in this condition till the evening of January 14, when the liver enlarged and signs of fluid appeared in the abdomen. To-day the pain is more marked on the right side than the left. He points to the right half of the abdomen from the costal margin to the iliac fossa and says he has pain in the right loins. The superficial tenderness extends from roughly the level of the seventh rib on the right side down to a point drawn from about the fourth lumbar spine forwards to a point about three inches below the umbilicus (seventh, eighth, ninth and tenth dorsal segmental areas). On his left side more or less tenderness is present over the same areas, but it is less well marked.

He now has tenderness of the scalp on both sides over the temporal, vertical, parietal areas, and over the right occipital area. This scalp tenderness is somewhat more marked on the right side than on the left.

Cardiac impulse very feeble. Cardiac dulness now extends 2½ inches (6·5 cm.) to the right of the middle line of the chest and 4½ inches to the left.

Over the apex, first sound weak, second reduplicated. No murmur of any kind can be heard. *Over pulmonary area*, second sound is scarcely heard. *Over aortic area*, sounds are extremely feeble.

Pulse about 76, very irregular, and with very small beat. Pulsation in the veins of the neck. Liver distinctly felt below the right costal margin. Signs of some fluid in the abdomen. Considerable quantity of æreated mucous expectoration, some of which is, however, browner than the rest. Cannot lie down in bed. Some crackling râles at both bases. No dulness.

Died January 17, 1895. The *post-mortem* revealed the following condition in the heart:—Right auricle somewhat dilated; right ventricle considerably dilated; left auricle much dilated, wall tough and fibrous; muscular substance diminished, excepting round the base of the appendix where it is hypertrophied; wall of left ventricle hypertrophied, but not greatly so; tricuspid valve dilated, smooth, no vegetations. From the auricular aspect the mitral valve appears as a long buttonhole slit with the edges in apposition throughout. It is very rigid. Fine bead-like vegetations on the free surface of the valve. The diameter of the valve measured with the cone is 0·8 inches (2 cm.). From the ventricular aspect the valve appears as a thickened, continuous ring of smaller diameter than from the auricular aspect. No marked change in aortic or pulmonary semi-lunar valves.

No micro-organisms discovered in the vegetations on the valve. Lungs congested. No infarcts, no consolidation. Liver, 58 ozs.; typical nutmeg.

Now the position of this referred pain and tenderness corresponds with that in affections of the stomach. We are thus brought face to face with the difficulty that the same areas may be affected in painful states due to lesions of the mitral valve and disturbances of the stomach. It might quite justly be urged that the presence of this pain and tenderness was due solely to a concomitant gastric disturbance; for troubles of digestion are not infrequent in these cases, and lend support to this objection.

I am unable to definitely prove my contention that this pain and tenderness is referred from the heart or disprove the objection that they are due to the stomach, but I think that a consideration of the following points tends to show that, in the majority of these cases, this pain and tenderness is of cardiac, rather than of gastric origin.

(1) True gastric disturbances are more common when the tricuspid valve fails and stagnation takes place in the system of the inferior vena cava. But it is exactly such a condition in which the left-sided pain and tenderness I have described is absent.

(2) In most cases where the first sound is abolished by a systolic murmur that is widely conducted referred pain is absent. But such cases not infrequently exhibit troubles of digestion.

Moreover, if the left ventricle fails, the first sound which was previously short and sharp may disappear, and a widespread systolic murmur be heard; at the same time, if a presystolic murmur was previously audible this murmur disappears. Under such conditions referred pain and tenderness may disappear to reappear again when the first sound returns under treatment. To illustrate this point I give the following case in which I twice watched this disappearance and reappearance of the first sound at the apex:—

Case 54.—To illustrate the disappearance of the referred pain, with alteration of the physical signs, and its reappearance when the physical signs improved again.—Rachel R. (V.P.H., Dr. Heron), aged 38, housewife.

First attack of St. Vitus's dance at the age of 13, second attack at 17. In 1875, at the age of 19, had an attack of acute rheumatism.

Since that time her heart used to beat quickly when she hurried, but she did her housework, and bore seven living children without complication.

1890.—She got a bad cough, and became very short of breath. Admitted to V.P.H. (Dr. Smith); went out well.

1892.—She became pregnant, and at the end of the fifth month came into V.P.H. again, with failure of the heart. Child born August, 1893, and three weeks afterwards again came into the hospital. From this time my observations on this case begin. She had evidently suffered for years from an ordinary type of mitral stenosis, and the least diminution in her general health would cause the left ventricle to fail.

September 4, 1894.—She had been admitted three weeks before with a systolic murmur at the apex, abolishing the first sound; the apical second sound was weak and reduplicated, and the pulmonary second sound was accentuated and single. There was no œdema of any part. She recovered in her usual manner.

August 30.—Menstruation began, and was accompanied by a good deal of pain in the lower abdomen and back. September 2. She was allowed up for half the day.

To-day there is a very marked change in her condition. She is taking no digitalis, so that this source of error is avoided, but menstruation still continues. Ears and lips are somewhat blue. No jaundice. No œdema anywhere. She has no pain, no headache, and no superficial tendency anywhere, but she complains that she feels faint and sick. She lies propped up in bed, but there are no marked objective signs of dyspnœa. Pulse small, and very irregular. Very slight pulsation of the veins in the neck. Apex beat in sixth space, five inches (12·5 cm) from mid-sternum. No thrill. Epigastric pulsation. Cardiac dulness begins above at the third rib, extends one and a half inches (4 cm.) to the right, and five and a half inches (14 cm.) to the left of the mid-sternum.

Over the apex, a loud systolic murmur is heard conducted into the axilla, but not heard at the angle of the scapula. Outside the nipple line and in the axilla the first sound is very feeble, but as we pass inwards there is a small area in the fourth and fifth spaces, extending for about one and a half inches (4 cm.) to the left of the left sternal edge, over which the first sound is short and sharp. The second sound is reduplicated. *Over the left second and third spaces* the second sound is accentuated, and not reduplicated. *Over the lower right sternal border* first sound is fairly well heard, and second sound is accentuated and single. Beyond a few crackling sounds at both bases, which were always audible in this case, there were no abnormal signs in the lungs. Tongue clean and moist. Nausea, but no vomiting. Complete loss of appetite. Bowels opened. Liver not enlarged. Urine

thirty ounces in twenty-four hours; no albumen. She was kept strictly to bed, and given tinct. strophanthus ηv., with extr. convallaria gr. v., every six hours. Menstruation ceased on September 9.

September 18.—She has greatly improved. The feeling of faintness, sinking, and giddiness is passing away. She has begun to take food well. Pulse now 88, irregular in force and rate, but more so in force than rate. Tension now maintained. Apex beat four inches from middle line, and cardiac dulness extends one and a quarter inches to the right and four and a quarter inches (11·25 cm.) to the left of the middle line of the sternum.

Over lower part of left back and in left axilla a loud, sharp, accentuated first sound is audible, but no murmur. *Over the apex* the first sound is still audible, but is obscured by a short systolic murmur. At the line of the anterior axillary fold both the sharp first sound, so well heard in the axilla, and the systolic, heard over the apex, are audible together. Second sound over the apex reduplicated. *Over the left second and third spaces* second sound accentuated and single.

By September 15 the pulse was nearly regular, and a typical short, sharp, accentuated first sound was now heard over the whole area from the left sternal border to the angle of the left scapula behind. Over the apex beat it was accompanied by a short systolic murmur. No murmurs within the ventricular diastole heard anywhere.

September 18.—All the faint, sinking feeling, the feeling of sickness, and the shortness of breath have been absent for some time, and have not reappeared. To-day pain has begun to appear again, and she is complaining of pain over the lower ribs under both breasts and under the scapula, more marked on the left side than the right. There is definite superficial tenderness over portions of the seventh and eighth dorsal areas on both sides. Tongue clean. Appetite good. Bowels opened daily. Pulse 90, somewhat irregular in rate. Tension maintained. The cardiac signs are as on September 13, excepting that a præsystolic murmur of short duration now precedes the short, sharp, accentuated first sound. The systolic murmur is heard over the front of the chest only, and is not conducted into the axilla or to the angle of the left scapula. At the angle of the scapula the first sound is distinctly heard. From this point onwards a præsystolic or diastolic murmur was audible, in addition to the local systolic murmur.

September 22.—She went home, engaged in house work, &c., and on September 27 came back in the same condition as on September 4. No sign of a præsystolic murmur; the apical first sound is entirely abolished at apex, in axilla, and at angle of left scapula. All the sinking faintness had come back, and she had orthopnœa as before; but all pain and superficial tenderness were absent.

(3) The referred pain and tenderness I have described may make their appearance quite apart from food, when the patient first gets up after a long stay in bed, or after some special exertion. This is best seen in those cases where pain and tenderness are not a marked feature. Case No. 47 showed this point well.

(4) If the pain is present it may be aggravated by food, but it may entirely disappear by rest in bed, or by other treatment directed towards the heart, although the diet remains quite unchanged.

(5) I think that the pain over the shoulder joint and in the left side of the neck (accompanied by superficial tenderness over the fourth cervical segmental area) which in gastric disturbances is so commonly associated with pain and tenderness within the areas we are considering is, as a rule, absent in lesions of the mitral valve. Thus, although the pain under the heart at the epigastrium, and in the region of the shoulder-blade is shared by both disease of the stomach and certain lesions of the mitral valve, the pain and tenderness over the shoulder-joint is not common to these two diseases.

The value of these considerations can only be gauged by repeated observations of cases of mitral disease; but, so far as my experience goes at present, I cannot help believing that this referred pain is due, under the conditions I have described, to the cardiac condition, and not to the stomach.

§ 6. *Referred Pain due to Enlargement of the Liver produced by Failure of the Right Side of the Heart.*

As a rule, no referred pain can be definitely associated with the right side of the heart. For tricuspid regurgitation is apparently unaccompanied by referred pain and tricuspid stenosis is of such rarity that no one physician can obtain sufficient cases for observation.

But when the tricuspid valve yields, an exceedingly important focus of referred pain not uncommonly makes its appearance in consequence of the distension of the liver. All cases do not show this referred pain, but it most often appears when the liver enlarges for the first time, especially if this enlargement takes place with considerable rapidity.

If the liver remains enlarged the pain may entirely disappear in a few days, and if we have reason to think that the liver is hard and fibrotic, or, if enlargement has frequently occurred before, it may be completely painless.

When the liver suddenly enlarges for the first time, the patient complains of pain over the right hypochondrium, iliac fossa, and in his right loin.

If pressure is made over the enlarged liver the patient complains of pain, and the liver is therefore said to be tender. Now it is quite probable that the liver itself is really tender, but in the large number of cases the supposed tenderness of the liver is found to be in reality superficial. Thus the tenderness is well marked in the right loin and when tested in the usual way the superficial structures of the abdominal wall and back are found to be tender. If well developed, the eighth, ninth, and tenth dorsal segmental areas can be marked out. The important fact in such cases is the presence of the tenth dorsal area. For the presence of the eight and ninth dorsal areas on the right side may be due to the occurrence of doubling from the left side, and may be simply a sign that the pain and tenderness from some cardiac lesion on the left side has become very intense. But no cardiac lesion causes primary tenderness between the level of the umbilical line, and, therefore, the presence of the tenth dorsal area which lies below this line, points in cardiac cases directly to the liver.

The same area is also usually present on the left side of the abdomen and back, but the tenderness is less intense, more spotty in distribution, appears later, and disappears earlier than that on the right side.

With the appearance of this tenth dorsal area on the body tenderness usually appears over the occipital region of the scalp. But as the eighth and ninth dorsal areas are also present on the right side of the scalp we usually find that the headaches and tenderness of the scalp are distributed over the vertical parietal and occipital areas.

An exceeding good instance of the referred pain and superficial tenderness due to secondary enlargement of the liver is given in fig. 6.

The tenderness is, however, usually somewhat wider spread when the liver enlarges.

Case 55.—Sarah S. (Victoria Park, Dr. Eustace Smith), aged 25. Single.

July 1893.—Acute rheumatism. Remained in bed a month and when she got up found that her breath was very short.

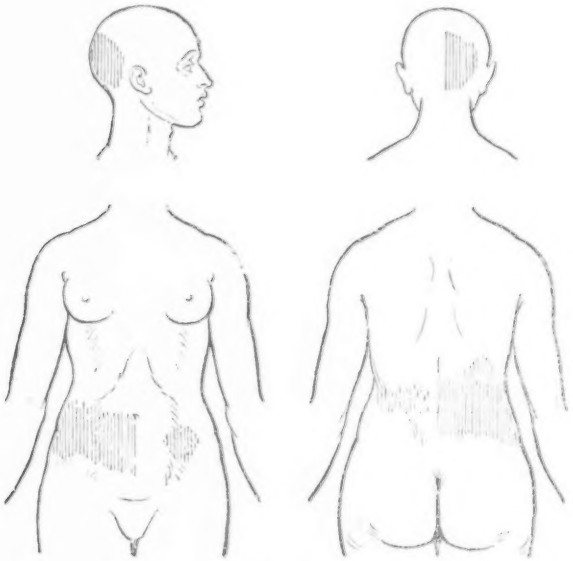


Fig. 6.

To illustrate the pain and tenderness over the 10th dorsal area, produced by Acute Enlargement of the Liver, in consequence of Cardiac Failure [Case No. 55].

Note the tenderness over the occipital region of the scalp.

September, 1893.—Admitted to V.P. Hospital with mitral stenosis and regurgitation.

Apex beat diffuse and forcible. Diastolic apical thrill. Upper limit, cardiac dulness third rib. Right limit, right edge of sternum. Left limit, nipple line. At apex first sound is loud and followed by a systolic murmur conducted as far as the left axilla. Second sound heard at the apex and followed by a short diastolic murmur. The pulmonary second sound was reduplicated. Aortic second sound normal.

Pulse small, irregular, easily compressible.

Edge of liver just felt.

She much improved, and was discharged on December 13, 1893.

As soon as she was discharged she tried to walk about the house, within a few days began to vomit. Much pain in the abdomen and headache.

Readmitted December 29, 1893. Condition as follows:—

Skin of face of a slightly yellowish tinge with a red spot on each cheek. Conjunctivæ not yellow. Ears and lips a little blue. No swelling of ankles. Distinct clubbing of the fingers. Knee-jerks normal. Plantar reflexes brisk.

She cannot lie flat in bed. Very short of breath on the least exertion. No fainting, but frequently feels faint.

She complains of pain in the abdomen below the navel and in the loins; she also has pain "across the shoulders and over the thorax below the level of the nipples." Much superficial tenderness.

Headache over the occipital region and over both temples associated with superficial tenderness of the scalp.

Pulse about 84, irregular both in force and rhythm, small stroke, compressible.

Distinct pulsation, but no marked distension of the veins of the neck.

Apex beat in fifth space in the nipple line. Pulsation also seen in third and fourth spaces internal to nipple line. Upper limit of cardiac dulness third rib. Right limit, cardiac dulness right edge of sternum. Left limit, cardiac dulness outside the nipple line. Heart's action very irregular. With the short beats a systolic murmur only is heard, but when the diastola is prolonged a soft diastolic murmur is heard. The first sound is feeble at the apex, but can be heard independently of the systolic murmur. The systolic murmur is conducted to the angle of the left scapula. No murmur heard over aortic area. First sound feeble over the right edge of the sternum.

Liver a good 2 inches (5 cm.) below the border of the ribs.

Suspicion of fluid in the abdomen.

Respiration 22. Slight cough; worse at night.

Some crackling sounds heard at the bases of both lungs and at apex of right lung.

Tongue red and a little furred. Bowels opened daily. She frequently vomited undigested food.

She has not menstruated for five months. Usually lasts two days. No pain.

January 9.—The feet are swollen to-day and there are distinct signs of fluid in the abdomen.

The liver edge can now be felt just above the level of the umbilicus.

She complains of pain in the abdomen below the umbilicus and occipital headache.

Marked superficial tenderness of the abdominal wall over the area in fig. 6, with definite tenderness of the occipital region of the scalp.

The liver continued to enlarge and the pulse became very irregular and small. She died on January 20, 1894.

On January 6 the temperature began to rise and oscillated irregularly between 100° and 98° F. (37·8° and 36·7° C.)

The urine was acid, sp. gr. 1025, and contained an amount of albumen which varied from about $\frac{1}{12}$ to $\frac{1}{15}$.

Post-mortem. — No pericarditis. Right side of the heart dilated. Tricuspid orifice admitted four fingers. Pulmonary valves otherwise normal. Left ventricle hypertrophied. Some thickening of the edges of the mitral valve which was somewhat stenosed. The valve was incompetent. Around its base on the auricular surface was a small layer of bead-like vegetations. The aortic valves showed fresh vegetations, which contained streptococci in considerable numbers.

No peritonitis. Fluid in the abdomen.

Liver weighed three pounds six ounces. Congested and fibrotic.

Kidneys congested, but otherwise normal.

Spleen small and congested with small fresh infarct in upper part.

Left lung healthy except for congestion at the base. Right lung congested. In middle lobe was a small infarct with some collapse around it.

Pelvic organs normal. Ovaries showed no signs of recent menstruation.

§ 7. *Paroxysmal Pain of Cardiac Origin.*

Writers on cardiac diseases express the most diverse opinions in their attempts to define the disease "angina pectoris," but are practically unanimous with regard to the symptoms of an "anginal" attack. Given the symptoms in any one case, the one group of observers refuse to diagnose angina pectoris if the physical signs of cardiac disease are present; whilst, at the opposite pole, are those who speak of angina pectoris whenever the symptoms are present, regardless of the age of the patient or the physical signs of disease in the heart.

But if differences of opinion are found between the various authorities during the life of the patient, still greater differences manifest themselves at the autopsy. Some cling to one anatomical lesion, some to another, and every case

that does not show a particular lesion is spoken of as false, or pseudo-angina pectoris.

From the point of view of the nomenclature of disease it is of obvious utility to confine the name angina pectoris to cases of a certain clinical type (*e.g.*, the classical cases in elderly men), or, to those which show some simple anatomical lesion (*e.g.*, occlusion of the coronary arteries.)

But throughout this study I am not engaged in a description of diseases, but in a physiological investigation of pain by the help of cases of disease. In this attempt to unravel the origin of pain it is necessary to look to physiological causes, and not to those descriptive characteristics which would enable a naturalist to separate one species from another. For the same anatomical lesion may produce different physiological effects, and the same physiological effects depend upon many different anatomical lesions. Thus, for my purpose, it would be a grave mistake to confine this section to a study of only one clinical class of paroxysmal cardiac pain. And yet I should be the last to attempt to bring all the cases here considered under the heading of a single disease.

In those cases, therefore, in which I am obliged to call the case by the name of some disease, I shall speak of primary angina pectoris where the symptoms are present without gross physical signs within the heart, and secondary angina pectoris where physical signs exist pointing to lesions of the aorta, or its valves.

Now, the symptoms of an anginal attack may be grouped under three main headings.

(1) A feeling of suffocation, accompanied by a pain at the epigastrium. This pain is variously described as "pressing pain," "bursting pain," "like being tied in with a cord," "as if the heart was too big."

(2) A sense of impending death. The face is said to become cold, the jaws stiff, and then this cold, "deathly" feeling spreads over the body.

(3) Violent radiating pains mostly over the upper part of the chest, and in one or both arms. These are accompanied by marked and wide-spread superficial tenderness, and are thus truly referred.

In any individual case these three symptoms may be present to a very varying degree. Thus my first illustration (Case 56) is chosen because of the complete development of these three groups of symptoms in the attacks. Yet for the purposes of classification of disease this man was probably a case of angina pectoris secondary to aneurism of the aorta.

Case 56.—*To illustrate the complete phenomena of an anginal attack in a man with aneurism of the aorta.* William D. (V.P.H. Dr. Eustace Smith), aged 38, railway porter.

At the end of 1892 he was out of sorts, and found he could not lift such heavy weights as before. One morning in January, 1893, he had a feeling of suffocation and giddiness, and his heart began to beat very fast. He felt faint and had to sit down. The doctor said he had "valvular disease." Throughout the next six months he had frequent attacks, in which he had a sudden feeling of suffocation with a choking sensation in the throat, and accompanied by a pain in the epigastrium as if a cord was pressing it in. In June, 1894, his attacks altered in character, and became like those seen in the hospital. These attacks came on particularly during his work.

He drank heavily till three years ago, and has been occupied with exceedingly heavy lifting work as porter at a goods' station. Denies syphilis.

On admission (October 1, 1894). Largely built man of 38. Face of a uniform pinkish-yellow colour. No anæmia. No cyanosis. No jaundice. No œdema. Exceedingly well nourished. Looks as if he had gained weight.

Pulse 90, regular, somewhat high tension. Arterial wall a little hard for a man of 38. Pulses equal.

No capillary pulsation.

No venous pulsation.

Heart's apex beat very feeble, and cannot be definitely localised. No pulsations over the upper part of the chest. No thrill.

Cardiac dulness begins at third rib, extends $\frac{3}{4}$ in. (2 cm.) to the right of the middle line of the sternum and $5\frac{1}{2}$ in. (14 cm.) to the left of the mid-sternal line.

Over the upper part of the sternum, and in the second right interspace, a sharp and ringing second sound is heard. The first sound is inaudible; and a distinct systolic murmur is heard over the upper part of the sternum to the aortic area. It is not conducted over the apex; the first sound is very short and wanting in tone, and closely resembles the second sound, so that the sounds at the apex have a "tick tack" character closely resembling that of a

fœtal heart. No murmur heard at the apex, or at the angle of the left scapula.

No cough or expectoration. He is short of breath on exertion, and cannot sleep flat upon the back.

No abnormal physical signs in the lungs.

Tongue moist and a little grey. Appetite poor. No vomiting. Bowels confined. Does not usually suffer from flatus.

Liver and spleen not free.

The pupils are equal to react to light and accommodation. The left does not dilate quite so well to shade as the right.

Urine normal.

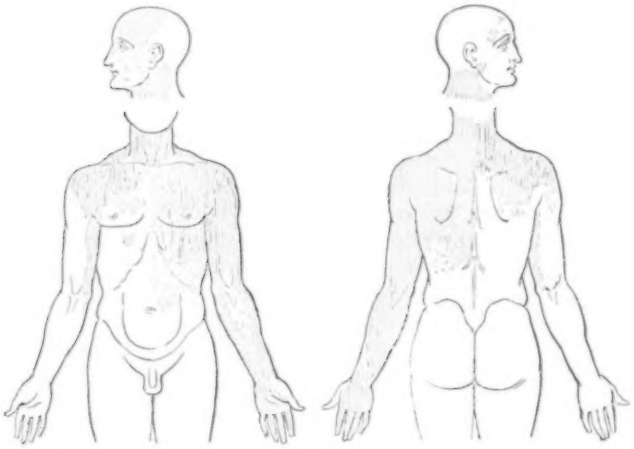


FIG. 7.

To show the widespread tenderness present after a major anginal attack in Case 56.

Nature of the Attacks and of the Pain.—On October 7 he was completely free from pain. During the night, at 1 a.m., on October 8, a patient in the ward became violent. William D. was sitting up to watch the struggle when he was noticed to lie back with his head thrown back over the pile of pillows, and his neck extended. His face was sweating, and he said he had a cold, dead feeling over the forehead, nose, and lips, which spread all over his face. The jaws felt stiff. He felt exactly as if he were dying. As he lay back he had a feeling over the region of the apex beat, and in the epigastrium, as if something were getting tighter and tighter. Then a shooting, tearing pain came on over the second and third interspaces, rapidly followed by a piercing pain over the anterior and posterior aspects of the left shoulder joint. Then the pain ran

over the left sterno-mastoid to the occipital region, and down the left arm to the top of the little finger. At the same time he suffered from marked headache over the left half of the forehead. During the later part of the attack, his heart was beating rapidly, but was not irregular. The attack lasted two minutes. It was followed by considerable mental depression, and he said he felt utterly exhausted. He complained of widespread soreness, and said he could scarcely bear his shirt, owing to the tenderness of his chest and back. (Fig. 7.) About a quarter of an hour after the attack he passed a considerable quantity of pale urine.

This represents a complete, fully-developed attack, characterised by the following symptoms:—(1) feeling of coldness; (2)

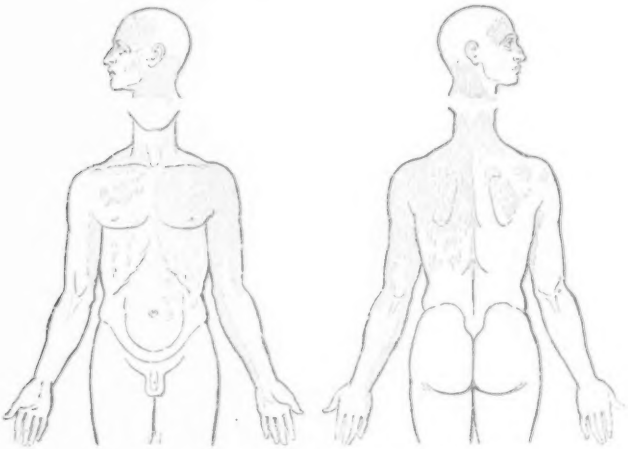


FIG. 8.

To show the tenderness present after an attack of paroxysmal pain [minor attack of the first type].

feeling of death and suffocation; (3) local bursting feeling at the heart; (4) violent shooting referred pains in chest and head, followed by intense widespread superficial tenderness. But during a long period of observation it became plain that he also suffered from minor attacks of two separate types.

First type (October 21, 1894). He had not slept well on account of pain in the upper part of left side of chest and back. In the morning his chest ached and felt sore. At 10 a.m. the pain began to shoot, and to spread as in a major attack, but it was of much less intensity. This shooting gradually ceased, and left behind it a sore aching feeling wider spread and more intense than before. Superficial tenderness was present on the left side of the

trunk, from the level of the jaw to the horizontal level of the tenth dorsal spine. (Fig. 8.) It extended down the ulnar border of the arm to the little finger. Some tenderness, but to a much less degree, was present on the right side of the chest. The whole fronto-temporal, mid-orbital, and frontal and nasal areas of the scalp and face were tender. There was no feeling of death or suffocation, and no sensation in the throat. This type of attack occurred when he was recovering under rest in bed and treatment with nitro-glycerine.

Second type (March 12, 1896). He woke feeling quite well. Whilst washing himself he raised both hands to the top of his head, and immediately sank back on to his pillows with a feeling of tightness across the chest. He felt suffocated, as if he would never get his breath again, with a cramped feeling in the throat as if something was being tightened. He felt a rolling, fluttering, irregular movement at the heart. It passed off, but returned again in a few minutes, and for an hour and a half he continued to have repeated small attacks of this nature. Before they began he was completely free from pain, but throughout the hour and a half during which they lasted a feeling of soreness over the upper part of the left chest and in the left arm gradually developed. There was no shooting or stabbing pain, as in the major attack, but a slow increase, with each little attack, of the sore aching pain. Finally he showed exceedingly marked superficial tenderness of the left side of the chest and back, from the level of the jaw to the horizontal level of about the tenth spine, extending down the left arm. There was very slight tenderness on the right side of the chest. Marked tenderness of the anterior part of the scalp. This type of attack seemed to occur when he returned to work after the major attacks had been abolished for a period of many months by treatment.

March, 1896.—The physical signs are still exactly as on admission in October, 1894, except that the systolic murmur is somewhat less marked.

Now this man not only had many major attacks, demonstrating all the symptoms of angina pectoris, but he also had minor attacks of two distinct types of much importance in attempting to analyse the phenomena of a major attack.

The one type was always preceded by some referred pain in the arm and shoulder. The attack consisted in the rapid spread of this pain to other parts. The heart did not become irregular. The sense of impending death was absent.

The second type came on after a definite exertion, and consisted of small exacerbations in a condition which was

present for an hour or more. He felt a tightness across the chest, a cramped feeling in the throat, and a rolling, irregular feeling at the heart. The heart seems to beat irregularly. These sensations lasted almost two minutes, leaving him with an uncomfortable feeling as if he would drop, and then recurred again and again at short intervals. On one occasion the whole state lasted one hour and a half. When these attacks begin there is no pain, but referred pain slowly appears, and becomes more marked after each little attack of suffocation. Finally, at the end of a considerable period, the pain and superficial tenderness become more marked. Thus the main feature of this type of attack is the suffocative feeling, and referred pain is not developed until these attacks are repeated many times.

Now just as this case showed minor attacks of two separate types, so we find that cases of primary and secondary angina pectoris range themselves at a point somewhere between two extreme types. On the one extreme lies the type in which the feeling of suffocation or strangulation and the sense of impending dissolution predominate. Referred pain may actually be absent, or if it is present, it simply follows the cardiac disturbance exactly as we should expect. On the other extreme lies the type that usually suffers from more or less pain and superficial tenderness apart from the attack. Here the spread of the referred pain and its increase in intensity are the main features of the attack. The heart's action is at first undisturbed, but as the pain continues to spread the heart begins to beat, not only with great rapidity but with increased force. The sense of suffocation and impending death may be absent.

Case 57 more nearly approached the first extreme type. He was an old man with profoundly degenerate arteries. The sounds of the heart were exceedingly feeble, and a musical systolic murmur was audible, apparently originating at the aortic valves. The attacks occurred in groups, and consisted firstly of palpitation and a sense that he was dying, and then secondarily of pain. The superficial tenderness before the first recognised attack was slight, but after the attack was over it had spread widely.

Case 57.—*To illustrate the type of anginal attacks, in which the suffocation and sense of impending death are the main features, and referred pain and tenderness a secondary phenomenon.*—John K. (V.P.H., Dr. Eustace Smith), aged 64; a canvasser.

In 1888 he began to suffer from palpitation of the heart. In 1889 he was in the hospital; but at that time suffered from no paroxysmal attacks. In 1890 he began to have slight attacks in which he felt faint. They began with gasping; he then felt as if he was going to die. Pain was not a marked feature of these attacks. In November, 1893, pain became a marked feature of the attacks, which then assumed the form described below.

He drank heavily as a young man, but has been a teetotaler for ten years. Has had many accidents, but no diseases, excepting one attack of gout in the left toe.

On admission. Medium-sized man of 64; looks considerably older. Marked arcus senilis. No cyanosis. No jaundice. No œdema. No wasting.

He has never fainted. On exertion he suffers from palpitation, which he says is quite different from the palpitation of the attacks. Not very short of breath, excepting during the attacks. Sleeps with two pillows, but does not need to be otherwise propped up.

Pulse 72, regular; of increased tension. The arterial wall is excessively tortuous and rough to the feel. Every artery of any size is tortuous and visible. The pulses are equal.

No capillary pulsation.

No venous pulsation.

Heart's apex beat seen and felt in fifth space ½ inch (1·5 cm.) outside nipple line. Not heaving. No thrill. No other pulsation visible in chest.

Cardiac dulness much diminished by emphysema. All over the front of the chest, within the areas enclosed by the right and the left nipple line is heard a high pitched, exceedingly musical systolic murmur. It is not conducted to the angle of the left scapula. Over the aortic area both first and second sounds are inaudible, whilst over the apex beat the first sound is not heard, but the second is occasionally very feebly audible. The weakness of the sounds of the heart was a very noticeable feature of this case.

Respiration 20: with the expiratory period prolonged. Marked elevation of the chest. Alæ nasi quiet. Single, non-paroxysmal cough; not very troublesome now. Very small quantity of grey mucous expectoration.

Physical signs in chest those of emphysema, but none of bronchitis.

Tongue clean, moist. No vomiting.

Liver and spleen not enlarged.

Urine 1010, acid. No albumen or sugar.

Nature of the pain and of the paroxysmal attacks.—The following account of an attack seen on February 24 is typical

of all his present attacks. In the morning he complained that he had slept badly, and felt a good deal of soreness in his chest. (Fig. 9). There were several spots of superficial tenderness confined to the left side of chest and inner aspect of left elbow. He had no headache, but there was some superficial tenderness above and around the left eye. At 10.30 p.m. he suddenly sat straight up in bed and said he was going to have an attack. His pulse was beating at the rate of 150 in the minute. He was sweating over the face, but there was no marked alteration of colour. Thirty seconds after the beginning of the attack he began to wheeze violently. In sixty seconds the pulse dropped in rate, and became very irregular. He complained that his heart felt as if

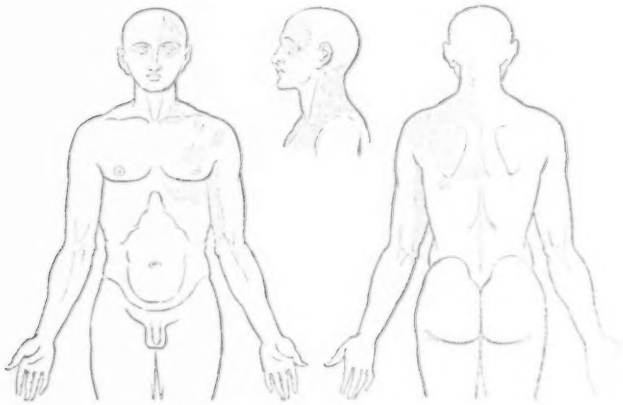


FIG. 9.

To show the tenderness which was present before the attack of angina pectoris in Case No. 57.

The dotted portions are those over which the tenderness was ill-defined and of no marked intensity.

it were swelled. The attack was over in two and a half minutes. He said that, when he called me, the palpitation had begun at the heart. Then the pain began to gather force. He had had pain in the chest and left arm before, and these pains rapidly increased, and were joined by a pain in the region of the hyoid. He complained that the skin of his forehead felt bulged, and that his nose felt swollen. Profound and widespread superficial tenderness of scalp, chest, and arms after the attack.

At 11 p.m., whilst I had my hand upon his pulse, he had a second attack. In the first ten seconds the heart beat 27 (at the rate of 162 in the minute). At the beginning of the second half minute it beat 24 in ten seconds (144 in the minute). At the

beginning of the third half minute it beat 14 in ten seconds (84 in the minute), but was very irregular both in rhythm and force. The pulse at the wrist was now very feeble and irregular. At the beginning of the fourth half minute it still beat 14 in ten seconds. At the end of the first half minute he began to wheeze violently and to clutch at his throat, complaining that he suffered pain there. The pain in the chest became much worse, and he felt as if he were dying. His body and head were sweating, and his hair became quite moist. He then gave a great yawn, and the attack was over.

He always insisted that the increase in the shoulder pain did not begin till after his attack was well on him; and that the attacks began with palpitation and were followed by pain.

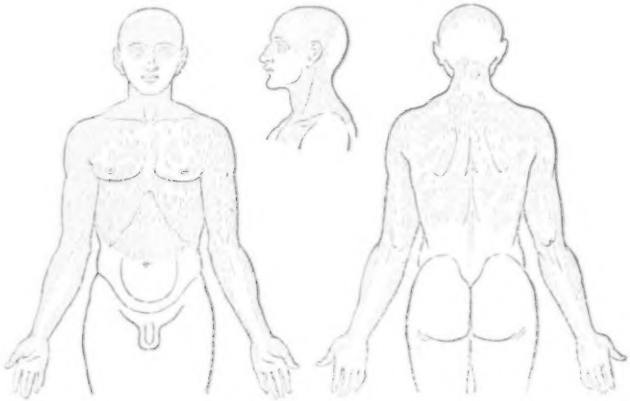


FIG. 10.

To show the extent of the superficial tenderness after the attack of angina pectoris in Case 57.

After the attack he had much widespread feeling of soreness of trunk and arms, and a feeling as if his head had swelled and tightened his scalp. Much widespread superficial tenderness of body and scalp. (Fig. 10.) The left arm felt numb, as if he had been lying on it. He could make no fine movements of his fingers, but there was no absolute paralysis. The loss of power seemed to him to be below the elbow. He seemed to be utterly exhausted, and was very depressed after the attack was over. Shortly afterwards he passed a considerable quantity of pale urine.

During the attack the systolic murmur was still heard, but neither first or second sound were audible anywhere. There were no moist sounds in the lung during the stage of wheezing.

These attacks enormously improved under treatment, and particularly with strict rest in bed. During the time he was in bed he had a few slight attacks, and it was very evident that the referred pain and superficial tenderness followed the attacks and were directly produced by them. For he would have an attack when all referred pain and superficial tenderness were absent, but after the attack marked referred pain and superficial tenderness would make their appearance mainly over the left side.

On the other hand, Case 58 more nearly approached the second extreme type. This case suffered from aortic regurgitation and stenosis as the sequel of acute rheumatism, and similar phenomena are found in some cases where the aortic regurgitation is of non-rheumatic origin. Here the attacks consisted mainly of a violent radiation of the referred pain present before the attack. An appreciable time elapsed between the beginning of the radiation of pain and the increase in the rapidity and force of the heart's beat.

It is interesting to notice that when the left ventricle failed after a series of attacks and a mitral regurgitant murmur became audible, abolishing the apical first sound, all attacks ceased. When the heart recovered the attacks returned. It is also interesting to note that this case died, not in an attack, but from cardiac failure.

Case 58.—*To illustrate an anginal attack in which the spread of referred pain was the primary phenomena.* Mary S. (V. P. H., Dr. Sainsbury), aged 18. In December, 1888, she had her first attack of rheumatic fever, which left her with an affection of the aortic valves. She remained fairly well, excepting that she suffered from pain in the chest and shortness of breath, until June, 1889, when she had a second attack of acute rheumatism. When she recovered from this second attack she had pain in the chest more or less continuously. Shortly afterwards, at 9 p.m. one night, she had her first attack.

From July, 1893, to March, 1894, she was under my observation day by day, and the following account is a *resumé* of the notes taken during this period by myself.

She is a finely-built rather stout girl of 18; looks a good deal older. Face pale, with occasional irregular flushes on cheeks and chin. No œdema; no jaundice; no cyanosis; no fainting; short of breath on exertion, and she occasionally suffered from periods during which she could only breathe when propped up. Palpitation on exertion and occasionally when lying in bed.

This palpitation always leaves her with a pain in the left breast and left temple, and if pain was present before, it is markedly increased by the palpitation.

Pulse, as a rule, about 80, regular, but easily runs up on exertion; typically low tension, with a strong stroke and marked regurgitant characteristics. Arterial pulsation visible in neck as high as the ears.

Extreme capillary pulsation; no venous pulsation. Cardiac pulsation visible over whole left half of chest. What appears to be the apex beat can be felt in the fifth space in the line of the anterior axillary fold. Cardiac dulness begins at second space above, extends to the right edge of the sternum and to a point to the left on the fifth rib in the line of the anterior axillary fold.

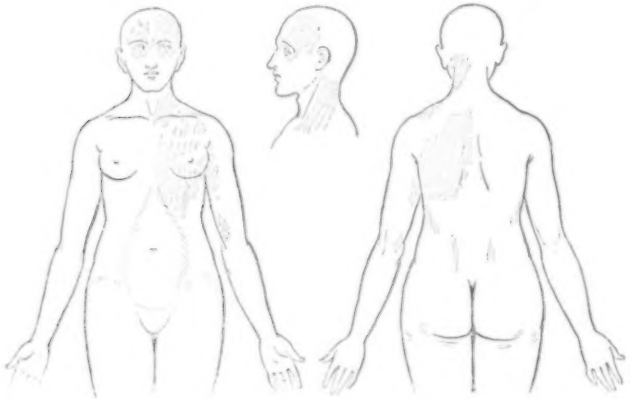


FIG. 11.

To show the extent of the superficial tenderness in Case No. 58, apart from an attack of angina pectoris.

Over the aortic area neither first nor second sound are heard but all over the sternum is a loud double aortic murmur (systolic and diastolic). Over the apex beat both first and second sounds are audible. The first sound is also heard at the angle of the left scapula. There is no mitral murmur either at the apex or angle of left scapula.

No abnormal physical signs in the lungs. Tongue clean; appetite bad. No pain after food; no vomiting; bowels somewhat confined. Liver and spleen not enlarged. Urine 1030 acid, no albumen, no sugar.

Nature and distribution of the pain and tenderness.—She usually complained of pain over the left breast and in the left interscapula region of the back. At times the pain was situated

over the inner side of the left arm. More or less superficial tenderness was usually present, usually on the left side, but sometimes, if the pain was severe, on both sides of the chest. The most common distribution of this tenderness was between the second and fifth rib in front, and from the level of the third dorsal to the seventh dorsal spine behind, and the tenderness involved either the upper third or the whole of the inner aspect of the arm to the elbow. (Fig. 11.) It was always most marked over the left half of the chest and back, and remained longer on the left side than the right.

The headache extended from the fronto-temporal region forwards over the eyes to a greater or less extent. This headache was accompanied by superficial tenderness of the forehead, and

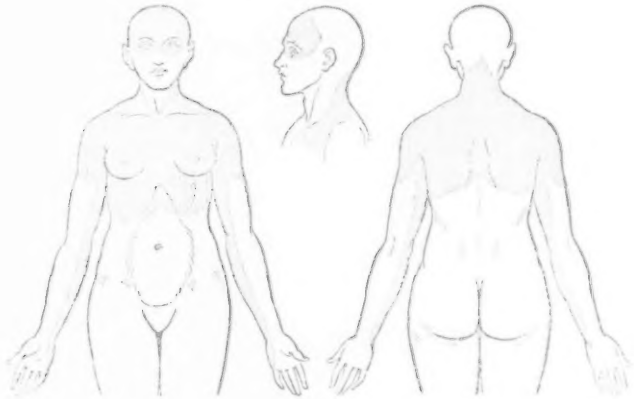


FIG. 12.

To show the extent of the superficial tenderness in case No. 58 during the time the pain was spreading, and 3 minutes before the quickening of the heart, in the attack of angina pectoris described in the text.

anterior portion of the temporal regions; it was usually bi-lateral, but occasionally unilateral confined to the left side. (Fig. 11.)

The attacks, which occurred at one time as often as once in twenty-four hours, were as follows: she suffered from pain for hours or even days before an attack. As a paroxysm came on, she would call out that she was going to have an attack. She said she felt the pain spreading. (Fig. 12.) She said it was going down the left arm and into the shoulder. The forehead was moist, the lips of a grey white, the area around the mouth pale, but the face still retained its pink colour. The pulse was 80. Then she called out that the pain was all over her chest. She threw herself backwards, arched her back, and clung to the rail at the head of the

bed. The forehead and cheeks flushed, and intense capillary pulsation was visible all over the face; the arteries of her neck pulsated intensely. The pulse ran up to 160, and became of extremely low tension. The heart beat seemed to shake the whole front of the chest. The arching of the back then ceased, the arms fell flat on the bed and she began to cough. The pulse rate rapidly fell. The attack occasionally ended in crying, and after it was over she generally passed a considerable quantity of pale urine of low specific gravity. It was followed by the most intense and wide-spread superficial tenderness of the thorax, back, arms, and scalp. The extent of the tenderness over the scalp and trunk closely resembled that shown on Fig. 10. The whole attack lasted about three minutes, but occasionally the first attack had scarcely passed off before a second came on.

As the pain spread it was noticed that the superficial tenderness became of much wider distribution. Thus I was, on more than one occasion, able to watch it spread down the inner side of the arm to the elbow, and then to the ulnar border of the hand. I also watched it spring into the neck and to the opposite side of the body. All this occurred before the pulse altered in rate. (Fig. 12.)

This I took to be an indication for treatment, and on several occasions, after the pain and tenderness had begun to spread, an injection of morphia or gr. xxx. of phenacetin prevented the great rise in pulse rate. The attack then completely altered in character. For the pain began to spread as usual, reached a certain point, accompanied by wide-spread superficial tenderness, and then slowly died away without the occurrence of the flushing, rapid pulse beat, or arching of the back.

During the attack she felt very faint, but the faintness was not sufficient to alarm her. She had no fear of impending death, although on more than one occasion the pulse became imperceptible at the wrist, and she seemed on the point of death.

The permanent pain, and the frequency of the attacks, were much lessened by treatment, and she left the hospital. I saw her three months later in a condition, shortly, as follows: the double aortic murmur was present as before, but the first sound at the apex was replaced by a systolic murmur, which was audible at the left angle of the scapula; the liver was distinctly enlarged; the mitral valve had evidently failed. She had had no attacks for three weeks, and she remained free from attacks for a further two weeks until the mitral murmur disappeared. With the marked improvement in the general condition of the left ventricle the anginal attacks returned.

She died in December, 1894, of cardiac failure.

To understand how severe attacks, like those of Case No. 58, may be developed in cases of aortic regurgitation, I give Case No. 59. Here the pain was, as a rule, not

paroxysmal, but closely resembled that usually seen in aortic disease, when unaccompanied by mitral regurgitation. However, under the influence of slight exertion, distinct paroxysms of slight intensity, but of the same nature as those of Case No. 58, appeared at infrequent intervals. Such cases are not uncommon; and are very instructive for the study of the origin of paroxysmal attacks arising in patients with organic disease of the heart.

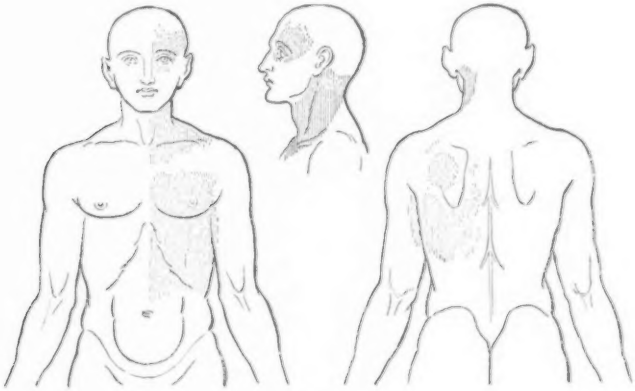


FIG. 13.

To show the extent of the superficial tenderness in Case No. 59, after the attack of "faintness" described in the text. Before the attack the tenderness was much less extensive.

Case 59.—To illustrate the origin of paroxysmal attacks of pain in cases of organic disease of the heart. Samuel F. (V.P.H. Dr. Eustace Smith), aged 40; labourer.

About Christmas, 1891, he was running to catch a train when he felt a pain "at the heart." This pain was exceedingly sharp, and forced him to stop. It never quite left him; and in April, 1893, it began to affect his left arm, but he continued with his work in spite of it until November, 1893. He was then seized with great shortness of breath, and suffered from much pain. A bad cough came on, and he spat up blood-stained phlegm. He was admitted to the hospital on December 11, 1893.

No history of acute rheumatism could be obtained, and he denies all venereal disease. His work is exceedingly heavy; and consists in unloading chests of oranges from the hold of a vessel,

and carrying them on his back up a flight of stairs into the warehouse.

On admission.—He is a well-built man of 40, and looks quite his age. No anæmia. No jaundice. No œdema. No clubbing of the fingers.

He fainted several times before admission, and within the first twenty-four hours after admission, felt faint more than once. He says his heart beats irregularly when he exerts himself. He is very short of breath when he walks about, but not when he lies in bed. He lies on the right side in bed on account of the pain in the left side.

He complained of pain over the inner side of the left arm as far as the elbows. Occasionally the pain runs down to the fingers of his left hand, and he has a stinging, numbing feeling in the middle, ring, and little fingers of the left hand. He also complained of pain at the epigastrium, and over points above and internal to the nipple, and under the outer part of the clavicle. All the pain was on the left side.

He also complained of headache in the left temple, extending over the left half of the forehead.

There was marked superficial tenderness over the chest, neck, temple, and forehead on the left side only.

Apex beat is feeble, but is felt in fifth space as far out as 2 in. (5 cm.) outside the left nipple line. No pulsation felt elsewhere in the chest.

The cardiac dulness begins above, at the third space, extends to the right sternal border, and to rather over 1 in. (2·5 cm.) outside the left nipple line.

Over the whole of the sternum, and over the apex, a loud aortic diastolic murmur is heard. No systolic murmur is audible. At the aortic cartilage the first and second sounds are not heard, whilst both sounds are distinct at the apex.

Pulse 100, regular, somewhat low tension. Pulses are equal on the two sides.

No cough or expectoration now.

No abnormal signs beyond a few *râles* at the right base.

Tongue a little furred. No vomiting now. No pain after food.

Liver and spleen not enlarged.

December 31.—He was free from pain until he got up to tea. At teatime he complained of a pain which started under the left breast and in the epigastrium. This pain then spread to the upper spaces, and then travelled slowly until it involved the left half of the neck and the inner side of the left arm. He then had a feeling as if he was going to drop. When I reached him he had crawled into bed. He was crying, and said he felt faint. The aching pain was still severe, but had ceased to spread. He was sweating profusely.

He showed exceedingly marked tenderness of the superficial structures of the chest, back, and neck, as in fig. 13. Headache

and tenderness were also present over the left temple and forehead.

There was no marked alteration in the physical signs, but the pulse was somewhat more rapid. The temperature had risen to 100° F. (37·8° C.)

He remained in bed for three weeks, and was treated with a pill containing extr. belladonna gr. ¼, pulv. opii gr. i. Although he had some occasional pain he had no further attacks.

Thus we must conclude that paroxysmal cardiac pain can be developed in one of two ways :—

(1) The pain is the expression of the cardiac disturbance and consists of a local epigastric pain, followed by more or less widespread referred pain. The attack is accompanied by a feeling of impending death and suffocation.

Attacks, more or less approaching this type, are best seen in men well on in life with calcareous arteries and feeble heart sounds.

(2) The pain is the main feature. The attack consists in the rapid radiation and increase in intensity of the referred pain that has been present before the paroxysm began. The feeling of suffocation or impending death may be absent.

Such attacks require a visceral lesion to produce the antecedent referred pain. They are, therefore, best seen in cases of aortic regurgitation, whether of rheumatic origin or not.

CHAPTER II.

THEORETICAL.

§ 1. *The Conditions within the Heart which lead to Referred Pain.*

Up to this point I have only attempted to show an association between certain physical signs and the presence or absence of referred pain. I shall now consider the significance of these signs, and attempt to show why they are associated with referred pain.

In an ordinary case there can be but little doubt about the presence of aortic stenosis and regurgitation. However, it

is not always so easy to be certain of the amount of the stenosis or the regurgitation. For it is only necessary to remind the reader that a systolic murmur over the aortic area, with the distribution of a murmur of aortic stenosis, may appear as a consequence of a roughening of the valves only, without any true stenosis. In the same way, a diastolic murmur, resembling the murmur of aortic regurgitation, may be present without abolishing the aortic second sound. In both these cases the pulse will help to show that but little stenosis is present in the one case, and little regurgitation in the other.

True aortic stenosis throws more work on the wall of the left ventricle during systole. The ventricle, therefore, hypertrophies, compensation takes place, and the patient suffers from no symptoms. Not so aortic regurgitation. For here the ventricle empties itself during systole, but as soon as the wall relaxes, at the beginning of the diastole, the blood pours back from the aorta and distends its cavity. In pure aortic stenosis the ventricle has to perform increased work during the systole, but is at rest during diastole. In pure regurgitation, on the other hand, there is not necessarily any increase in the work required during systole; but just at the moment when the wall is not toned to meet an effort, the blood comes pouring back into its cavity from the aorta. Now it is this distension, at the moment when the walls of the cavity are at rest, that is most pernicious, and it is this condition, above all others, that is most liable to lead to referred pain.

When both regurgitation and stenosis are present the condition is still worse. For not only has the systole to overcome a greater intra-ventricular resistance, but the diastole is broken by a back rush of blood from the aorta. The left ventricle hypertrophies to meet this increased resistance in front, but the increase in the force of the ventricular contraction will also increase the force with which the blood rushes back from the aorta at the beginning of the diastole. Thus referred pain is more likely to be present with aortic regurgitation than with stenosis, but most certainly when both lesions are combined.

But pressure within the cavity of the ventricle during the diastolic period gives rise to a second evil. The walls of the cavity are particularly liable to dilate, and the contents of the cavity to permanently increase. The ventricular wall now acts at a disadvantage on its contents, and they may possibly not be completely expelled during the systole. Thus the ventricle is never actually empty, but is always somewhat distended, an ideal condition for the production of referred pain. In most cases this is probably but a temporary condition, arising from some additional exertion in a heart normally working at the top of its power. So long as the patient remained more or less at rest, the ventricle is able to empty itself during systole; but it is working at the extreme limit of its power, and a little extra difficulty will render the complete emptying of the cavity at each systole impossible. In young people some excessive effort is necessary to produce this condition, but in older people, and especially in those cases where the lesion of the aortic valve is not of rheumatic origin, it may arise after any small exertion, such as getting up after a rest of a day or two in bed.

This permanent distension of the left ventricle cannot exist long, or to any very great extent, without the mitral valve becoming incompetent. Directly this happens the pressure within the ventricle becomes relieved, and the cavity is again emptied of blood at each systole. An enormous load is taken off the walls of the ventricle, for the blood can now leave its cavity by two roads. If the resistance in front is great, a considerable quantity of blood is now pumped, at each ventricular systole, into the pulmonary circulation, through the incompetent mitral valve, for the highest pressure in the lungs is low compared with that in the aorta. The aorta then receives less blood, the pressure within it falls, and the tension in front is relieved. Thus, as soon as the mitral valve yields, the permanent pressure within the left ventricle is relieved by a self-regulating mechanism, and referred pain disappears.

In the same way the essential conditions for the production of referred pain are absent in those cases where the

incompetence of the mitral valve arises coincidently with disease of the aortic valves during acute rheumatism.

Thus, whether the yielding of the mitral valve be a primary or a secondary occurrence, free regurgitation through this orifice acts as a safety vent to the ventricle, and prevents that distension of its cavity during diastole, necessary for the production of referred pain.

So far, the physical signs, upon which we have depended for a knowledge of the condition of the circulation in valvular disease, are fairly easy of interpretation. But a similar attempt to translate the physical signs produced by disease of the mitral valves, is beset with many difficulties. I will, therefore, begin with those cases where the interpretation of the physical signs presents least difficulty.

If a systolic murmur, audible at the apex and well conducted to the back, abolishes the first sound at the apex, in the axilla, and at the angle of the left scapula, and especially if the pulse is at the same time one of low tension, the lesion of the mitral valve is probably pure incompetence.

In such cases the blood is expelled by each contraction of the ventricle, not only into the aorta, but also into the auricle. A free path is open into the pulmonary circulation, and the pressure in the ventricle, therefore, always remains low. With each diastole the ventricle dilates, and the blood flows in again from the auricle at the pulmonary pressure. Thus the heart has to act more frequently to circulate the same amount of blood, and the pulse is increased in rate, but its tension is low. As extra work has to be done by the left ventricle, its wall hypertrophies, and complete compensation may occur. It is true the auricle receives blood from the left ventricle during the auricular diastole, but the blood does not enter at high pressure. Moreover, the left auricle can empty itself freely at each stroke through the free opening of the mitral valve, and thus the mean pressure within its cavity does not rise. Hence such cases of pure mitral regurgitation are the least serious of all cardiac lesions, and are unattended by referred pain.

Let us now consider the condition of the circulation in a pure case of mitral stenosis, characterised by a pre-systolic

murmur of the typical quality, terminated by a sharp first sound. It is almost universally acknowledged that this murmur is produced by a jet of blood forced through the narrowed mitral orifice by the sharp contraction of a well-acting and probably hypertrophied auricle. For it is the most capricious of all cardiac murmurs; it disappears as soon as the heart shows signs of failing, but reappears as the patient recovers. It is always absent some time before death. If absent when the patient is at rest, it reappears when, by any exertion, such as lifting the arms, extra work is thrown on the heart. The blood enters the auricle during the diastole. Then follows the auricular systole, which drives the blood forcibly through the narrowed orifice, producing the pre-systolic murmur. The ventricular systole follows immediately, and terminates abruptly the flow from the auricles. Then follows the auricular diastole, during which the cavity of the auricle can remain at rest, except for the somewhat high tension in the pulmonary circulation, which always follows a narrowing of the mitral valve.

Under such conditions the pressure in the auricle is high, but the contractile force of the auricle is increased to meet it; the diastole of the auricle is uninterrupted, and there is nothing to cause referred pain. It is, of course, possible that some exertion, which, though actually not great, is relatively considerable, owing to the diseased condition of the valves, may upset this circulation. Blood would then accumulate in the auricle, and it would remain permanently distended, and referred pain would result. This probably accounts for the occasional presence of referred pain in these pure cases of mitral stenosis. Pain is, however, the exception, and only occurs after some definite indiscretion, and not during the even tenour of a quiet life.

But imagine that just when the powerful auricular systole has expelled the blood through the narrowed mitral orifice, a strong jet of blood is forced back into its cavity, just as its walls are relaxing, we should then have exactly the condition which we found so fruitful a source of referred pain in aortic regurgitation, except that the auricle here

bears the brunt of the diastolic pressure instead of the ventricle.

This is the condition that I believe exists in those cases where an apical systolic murmur is present, accompanied by a short, sharp, accentuated first sound.

The actual mechanism of this short, sharp, first sound in mitral stenosis is still uncertain. But whether its origin be valvular or muscular, it seems to be associated with the contraction of a well-acting left ventricle on a light load. If the ventricle loses power, and dilates, this sharp first sound disappears. At the same time, the systolic murmur may become audible over the back and in the axilla. Thus, whatever the mechanism of this accentuated, sharp, first sound may be, it seems to be associated with a well-acting left ventricle and a stenosed mitral orifice.

It might be objected that in pure mitral regurgitation blood is also forced into the auricle at the moment of its diastole by the contraction of the left ventricle, and yet that this condition gives rise to no referred pain. But we must remember that in such cases the mitral orifice offers no obstruction. The contracting auricle can empty itself with ease into the ventricle, and the blood which regurgitates from the left ventricle passes easily into the pulmonary circulation. In mitral stenosis, on the other hand, before the left ventricle dilates and the heart fails, the tension in the pulmonary veins and left auricle are high, owing to the obstruction to the onward flow. This increase of tension is evident from the frequency of hæmophysis in cases of mitral stenosis. Out of twenty-five consecutive cases, eighteen suffered from hæmoptysis at one time or another. Thus even a slight increase in the auricular contents during its diastole, especially if it take place with rapidity and considerable force, must act injuriously.

The following considerations also show that the regurgitation in these cases of considerable mitral stenosis occurs at a considerable pressure. The pulse of mitral stenosis, at any rate in the earlier stages, is of well-maintained tension, though the stroke is poor. Now, before blood can leave the left ventricle, and pass into the aorta, the tension in the

aorta must be overcome. Thus the contents of the left ventricle must be under fair pressure. If regurgitation takes place from the left ventricle, this regurgitation must, then, also take place under fair pressure. On the other hand, the pulse of pure mitral regurgitation is distinctly one of low tension, and easily compressible. This points to the regurgitation through the mitral orifice also occurring at low pressure.

Thus, in conclusion, it would seem that the most potent conditions for the production of referred pain are: firstly, the maintenance of considerable tension within a cavity of the heart, accompanied by, secondly, a sudden accession of tension (owing to regurgitation) at the moment when the walls of that cavity are dilating after systole.

Throughout this paper I have omitted all consideration of complex combinations of valvular lesions. For, although such cases form interesting problems for solution, when the principles which govern the presence or absence of referred pain are once determined, the conditions of the circulation are too complicated to aid in the determination of such principles. Moreover, the various groups into which such cases fall, do not occur with sufficient frequency. I have also neglected an interesting group of cases where referred pain appears as the accompaniment of acute endocarditis, arising during the course of an attack of acute rheumatism.

§ 2. *The Sensory Supply of the Various Portions of the Heart.*

I have attempted to show, in the previous section, that the referred pain, which occurs in certain cases of mitral stenosis, is due to conditions of pressure within the left auricle. Now, on examining the records of such cases (p. 186 to 189), we find that the referred pain and superficial tenderness lie within the areas from the fifth to the ninth dorsal, but mainly within the sixth and seventh dorsal segments. Thus we may assume that if the explanation of the causes which give rise to referred pain in such cases is correct, the sensory impulses from the auricle pass into the

central nervous system at the level of the fifth, sixth, seventh, eighth, and possibly the ninth dorsal segments, but mainly at the level of the sixth and seventh dorsal.

In the same way the referred pain, in cases of aortic regurgitation, or of combined aortic regurgitation and stenosis, were attributed to abnormal pressure in the left ventricle. Now the pain and superficial tenderness in such cases lies mainly over the second, third, fourth and fifth dorsal, with occasional implication of the fourth cervical and sixth dorsal. Thus the impulses from the left ventricle probably pass into the cord at these levels.

A study of aneurisms of the aorta (p. 168) taught us that the referred pain and superficial tenderness they produce may be divided into three groups. Firstly, with aneurisms of the ascending arch, the pain and superficial tenderness lie over one or more of the following areas —first, second, third dorsal, third and fourth cervical. Secondly, if the transverse arch is implicated, the pain and tenderness may lie over the anterior surface of the neck, especially within the inferior laryngeal area. Thirdly, when we have reason to think that the aneurism has involved that part of the aorta which lies beyond the entrance of the ductus arteriosus, the pain and superficial tenderness may lie within the fifth, sixth and seventh dorsal segmental areas.

These results may be tabulated as follows :—

Sensory Supply of the Heart.

Transverse arch of aorta	...	Inferior laryngeal segment
Ascending arch of aorta	...	3rd and 4th cervical 1st, 2nd and 3rd dorsal [? 4th dorsal.]
Ventricle	...	2nd, 3rd, 4th and 5th dorsal [? 6th dorsal.]
Auricle	...	5th, 6th, 7th and 8th dorsal [? 9th dorsal.]

§ 3. *The Physiology of Cardiac Pain.*

Pain does not exist without a cause. It is simply the expression of the stimulation of sensory nerves within the heart, and this stimulation has a definite aim.

The first effect of the presence of pain will be to warn the patient against exertion. Under the influence of this warning the body is placed more at rest, and the heart will have a chance of recovering, in consequence of the diminished call made upon it. Pain, wherever it occurs in organic heart disease, is a warning and an intimation that rest is required.

But, probably, the stimulation of the sensory nerves, of which pain is the expression, performs a second function. We know that the depressor nerve conducts sensory impulses from the heart to the medulla, which cause dilatation of the visceral capillaries, and so lower the pressure in the distal portion of the vascular system. We also know that augmentor fibres pass from the dorsal cord to the heart, increasing not only the rate but the strength of its contractions. Now, these fibres have been found in the second, third, fourth, and fifth anterior roots, and it is quite possible that the sensory portion of the reflex arc is of much wider extent.

Thus it is possible that these pain impulses may be the expression of the afferent arc of one, or both, of these reflex mechanisms; at any rate, in those cases where the pain lies over the upper dorsal or third and fourth cervical areas.

It is, possible, therefore, that the implicated segments of the central nervous system are both acted on, and react upon the heart. This must be borne in mind during the study of paroxysmal attacks of referred pain. A very widespread disturbance of the heart, especially if it occur suddenly, will cause extremely widespread referred pain, owing to the intensity of the stimulus poured into the central nervous system. Before the disturbance in the heart's activity, no referred pain or tenderness will be present; but during and after the manifestation, both will be intense and widespread. This is the one extreme type of anginal attack. But, supposing pain impulses of considerable in-

tensity have been pouring into the segments of the central nervous system for a considerable time, the resistance within the nervous system may ultimately break down, and the impulses will spread up and down the cord. When the cord is widely implicated, especially if the impulses are of considerable intensity, the motor mechanism will become involved, and not only will the tension fall rapidly [depressor action], but the heart will beat with greater rapidity and force [augmentor action]. This is the second extreme type of anginal attack, in which the antecedent referred pain is the main feature, and the cardiac disturbance appears to be secondary to the spread of the pain.

But in order that the stimulation of this reflex arc may occur, the heart must not be worn out by disease of long existence. For, as I pointed out above, much referred pain may appear when the liver enlarges for the first time; but if it remains enlarged, or if the enlargement has occurred many times, the pain ceases to make its appearance, owing to the changes undergone by the liver substance. In the same way, so long as the condition of the heart remains fair, referred pain appears when the necessary conditions for its appearance are fulfilled, and the heart then reacts to the stimulus so produced. But, later on, towards the end of the case, the heart becomes worn out, and structural changes occur. Such hearts react neither to changes of internal pressure nor to drugs; and the patient passes away painlessly, and in spite of all our efforts.

§ 4. *The Distribution of Referred Pain and Tenderness in Diseases of the Heart considered from the Developmental Aspect.*

In section 2 of this chapter I attempted to map out the sensory supply of the heart, and the result must appear, at first sight, somewhat startling. For it would appear that the ascending arch of the aorta receives its innervation from the highest segments, whilst the impulses from the auricle enter the lowest segments that stand in relation to the heart. Thus the sensory visceral innervation of the heart is

upside down in relation to the present position of the adult human heart.

Now it is worth considering, shortly, what we know of the development of the heart in attempting to explain this remarkable fact.[1]

Let us take up the development of the heart at the stage where the two tubes have fused to form a single tubular heart. The hinder end is continuous with the two vitelline veins, whilst the anterior end bifurcates into two primitive aortæ. At this time, the heart lies in the middle line under the upper part of the alimentary canal, and clearly shows the division into three portions. The hindmost of these becomes the auricle, the middle portion the ventricles, and the most headward portion the bulbus aortæ, and ultimately the ascending arch of the aorta. This tube then becomes bent on itself, so that the hindmost, or auricular portion, comes to lie above the middle or ventricular portion.

Now the plan upon which the sensory supply of the sympathetic system to the viscera is laid down, seems to be very antique, and to represent an early stage of vertebrate phylogeny. The sensory supply of the heart bears out this statement, in that the impulses enter the segments of the central nervous system just as if the auricles were still the hindmost portion of the heart, and as if the heart were a median organ.

That portion of the aorta, from the valves to the innominate artery (ascending arch) is innervated like the ventricle, from the second, third, (and, perhaps, the fourth) dorsal segments, and also refers into the first dorsal, and third and fourth cervical areas. Thus the next segment, headward of the first dorsal, is the fourth cervical. The fifth, sixth, seventh, and eighth cervical are skipped, and a gap exists, as I pointed out in my two previous papers.

[1] The following considerations will be rendered clearer, to those of my readers who are not familiar with the development of the mammalian heart, by a reference to Figs. 176, 177, 178, 179, and 181 in Quain's "Anatomy," 10th edition, vol. i., part 1. The originals of these beautiful figures are found in His, "Anat. d. Mensch. Embryonen," a work that is in the hands of but few persons. I have, therefore, referred the reader to Prof. Schäfer's excellent modifications of the original figures.

Now the third and fourth cervical areas lie over a part of the neck, which is definitely somatic in origin, and has no connection with the structures formed from gill arches.[1] This area of skin grows forwards from the dorsal surface of the body, encroaching on the gill arches and their appendages. Now, as I pointed out before,[2] the sensory portion of the first cervical segment is contained in the trigeminal (fifth nerve), and the second cervical (whose sensory fibres enter in the cervical region at the proper level) receives its afferent impulses from the posterior part of the scalp. Thus the third cervical segment comes to be the highest segment which receives impulses from a true somatic skin surface, and is the uppermost segment which supplies true somatic viscera, *e.g.*, the ascending arch of the aorta, and the apex of the lung.

In front of the anterior border of each sterno-mastoid, lies a triangular area, which the dissector knows as the anterior triangle of the neck. Within this area referred pain and superficial tenderness appear, with lesions of such essentially branchial structures as the larynx and its appendages. The ventricles and the lung never primarily refer into this area. Thus, I pointed out that the areas within this region, like those of the jaws and face (excluding the nose, eyes, and forehead) belonged to a branchial system of segmentation, apart altogether from the somatic segmentation, and that of the areas on the forehead and scalp.

Let us turn for a moment to what we know of the development of the aorta in man and the higher vertebrates. The bulbus arteriosus is the most anterior or headward portion of the heart. It ends in front in a series of lateral vessels, which run from the ventral surface towards the back. The front four of these vessels run in the four branchial arches; of these the first three form the carotids and their branches, and do not concern us here. The fourth of these vessels, which runs in the fourth branchial bar, behind the

[1] *Cf.* Part II., BRAIN, 1894, p. 477; also His. "Anatomie Menschlicher Embryonen," Leipzig, 1885.

[2] BRAIN, 1894, p. 477.

third branchial cleft, forms the transverse arch of the aorta on the left side, and the innominate artery on the right side. The fifth vessel, which runs behind the fourth cleft, forms the pulmonary artery. On each side, dorsally, these five vessels are connected with one another. Thus, the fourth vessel, running in the fourth arch on the left side (the future aorta), unites, on the dorsal surface of the body, with the fifth vessel (the future pulmonary artery). The point at which this union takes place is marked on the adult aorta by the entry of the obliterated ductus arteriosus, which originally united the stem of the pulmonary artery with the aorta, and is the remains of the dorsal portion of the fifth branchial vessel. Thus we have now divided the aorta as we know it, in the adult with three portions: (1), from the semi-lunar valves to the level of the origin of the innominate. This is the representative in the adult of the bulbus arteriosus, the most headward division of the tubular heart. (2), from the innominate to the point of entry of the ductus arteriosus. This part represents, in the adult, the fourth branchial vessel. (3), all the aorta beyond this point—a true somatic dorsal vessel.

It is, therefore, peculiarly interesting to find that aneurisms of the ascending, or first part of the aorta, cause tenderness over the third and fourth cervical, and first, second, third dorsal segmental areas; all of which are purely somatic, and either identical with, or a continuation headwards of the areas to which the ventricle refers. On the other hand, aneurisms of the transverse arch, the remains of a true branchial vessel, cause pain and tenderness within the inferior laryngeal areas of the neck, in common with so truly a branchial organ as the larynx. This is the more instructive, from the fact that the larynx proper is developed from the structures of the fourth branchial bar, of which the transverse arch of the aorta represents the branchial vessel.

Much of what I have said in this section may be fanciful, and may be negatived by future investigation. But, at any rate, for the present, such embryological considerations seem to me to offer the only hypothesis to explain the distribution of referred pain and superficial tenderness in diseases of the heart and aorta.

CHAPTER III.

Pain in Diseases of the Respiratory Organs.

§ 1. *Character of the Pain.*

In my first paper I scarcely touched on the pain produced by diseases of the lungs, and neglected the pain which may occur in the course of phthisis; for whatever pain is present in this disease throws little light on the nature and causes of referred pain, and on the limits and anatomical distribution of areas of superficial tenderness. But although of but slight scientific interest for the determination of localisation, in the central nervous system, the distribution of pain and tenderness is of considerable practical importance, and occupies a considerable field in the clinical picture of many diseases of the lungs. The study of pain in its different forms in various diseases of the lung and pleura, also forms a useful demonstration of the methods by which widespread pain and large territories of superficial tenderness are to be interpreted.

Two quite distinct varieties of pain, accompanied by two even more distinct types of tenderness, may make their appearance in the course of diseases of the lung and pleura. Pain of the first type is local, and occupies no predetermined spots or areas. It is situated over the focus of disease, and usually in those intercostal spaces which cover a patch of pleural friction. It is accompanied by no true superficial tenderness, but marked deep tenderness is evoked by pressure or percussion. Pain of the second type is referred—that is to say, it is not necessarily situated over the focus of disease. It runs through or around the body, and has a focus behind and one in front. If severe, or of any duration, it is accompanied by more or less marked tenderness of the superficial structures of the chest, and this tenderness lies over predetermined spots or bands. Thus, referred pain and tenderness do not point directly, but only indirectly to the focus of disease.

(*A*) *Local Pain in Pulmonary Diseases.*

This type of pain occasionally appears in the neighbourhood of phthisical centres, or large areas of pneumonic consolidation; but in the large majority of cases it stands in definite relation to a pleuritic rub. It is said to be "stabbing" or "catching" in character. It is absent when the side is at rest, is intensified by each inspiration, and becomes agonising during the long and deep inspiration which follows a cough, a laugh, or a sneeze. It is relieved by any treatment that puts the side at complete rest, such as successful strapping. The patient describes the pain as like the sudden stab of a knife into the side. But this stab does not go through the chest from back to front, as is the case with severe referred pain, but is localised to one spot.

This pain is frequently associated with marked tenderness on pressure or percussion in the intercostal spaces.[1] Thus, by fairly heavy percussion, an area can generally be marked out within which deep tenderness exists. Now within this area pleuritic friction will usually be audible. I must warn the reader from supposing that referred pain and superficial tenderness never co-exist with an acute pleurisy. But where superficial tenderness, of the usual type that accompanies referred pain, exists, it is due to the disease of the lung, of which the pleurisy is a secondary phenomenon, and is not due directly to the pleurisy. For in a considerable number of cases of acute pleurisy all superficial tenderness is absent, and the presence of the patch of deep tenderness, corresponding more or less closely to the area of friction, can be easily demonstrated. Thus, before proceeding to test for deep tenderness, it is well to determine that all superficial tenderness is absent, or the results of the examination will be fallacious.

This deep tenderness, in most cases, seems to be definitely due to pressure upon the inflamed pleura. For it corresponds very fairly with the area of audible friction, and is less easily obtained over the ribs than over the inter-

[1] Walshe ("Diseases of Lungs," third edition, p. 269) recognised this tenderness on pressure.

costal spaces. It disappears with time, even though the friction may remain, and thus it is sometimes a useful guide to the duration of a friction sound, present when the patient first comes under observation. Local pain and deep tenderness usually disappear when serous fluid is effused; but I have more than once seen it persist over the upper part of the purulent effusion in empyema.

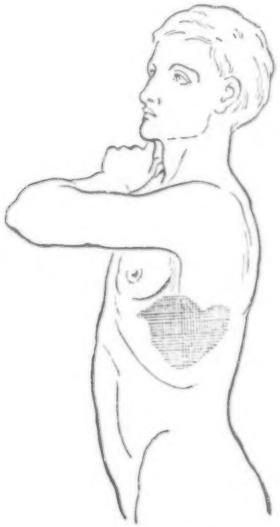


FIG. 14.

To show the area of deep tenderness evoked by pressure or percussion in a case of acute pleurisy [Case No. 60].

Case 60.—*To illustrate the local pain and tenderness on deep pressure which accompanies a pleurisy.* John K. (V.P.H., Dr. Heron); aged 44; coachbuilder.

In 1888 he is said to have suffered from pleurisy, but no detailed account of the attack can be obtained. Some cough each winter since then, but he has been able to do his work. Is a teetotaller. No family history of phthisis.

On admission (December 11, 1893).—Medium-sized well-built man of 44. Looks older. No marked anæmia. No wasting. No cyanosis. No night sweating.

Respiration, 24. No marked dyspnœa. Hard cough in paroyxsms, which seem to choke him. Expectorates mainly aerated mucus. No tubercle bacilli found in sputum.

He complains of pain in seventh, eighth, and ninth spaces in the left axilla. The pain is increased by taking a deep breath, and becomes exceedingly bad in a paroxysm of coughing.

No superficial tenderness, but marked tenderness on pressure or percussion over a patch the size of the palm of the hand in the seventh, eighth, and ninth spaces (fig. 14). No headache. No scalp tenderness.

Both sides of the chest move equally. Percussion note somewhat hyper-resonant. Breath sounds everywhere weak, but expiration is not prolonged. Vocal resonance everywhere feeble. A few scattered râles are heard over the lower part of the left lung.

In the left axilla marked rough friction is heard, extending over the seventh, eighth, and ninth spaces. The area within which friction is heard almost exactly corresponds to the area of deep tenderness (fig. 14).

No other signs or symptoms of importance. Temperature not raised.

December 26.—Friction is less marked. All pain has practically gone, except when he takes a very deep breath. The area of deep tenderness is contracting, and is less marked.

January 3.—All pain and deep tenderness gone.

Sometimes an infarct in the lung will cause a small patch of pleurisy if situated near the surface. Such cases form exquisite examples of the localising value of deep percussion tenderness. I have several times had the opportunity of verifying the position of the infarct which caused this local tenderness. In Case 61 both infarcts were diagnosed during life, and verified after death.

Case 61.—*To illustrate the deep tenderness sometimes present when infarcts of the lung cause small patches of pleurisy.* Emma D. (V.P.H., Dr. Harris); aged 47; nurse.

At the age of 14 years she had chorea, "due to a fright." It lasted fifteen months. At no time in her life has she had anything resembling acute rheumatism.

She had been working very hard as a sick nurse, and on December 10, 1893, began to feel very tired, hot, and feverish. December 11 her temperature was 104°. She was in bed for a fortnight, with a varying temperature, and then got up. January 10, 1896, the temperature rose again, and she felt very ill.

On admission (January 18, 1896).—She is a small, used-up looking woman of 47. Arcus senilis beginning to appear in both eyes. Face of a grey, dusky pallor.

Respiration rapid. Very short of breath. Some orthopnœa.

Directly after admission she began to complain of pain in the *right* axilla in the sixth, seventh, and eighth spaces. There was no superficial tenderness, but exceedingly marked deep tenderness in the sixth, seventh, and eighth spaces in the mid-axilla.

January 19.—The percussion note is diminished over the lower axilla on the right side, extending back to the line of the angle of the scapula. Over this area the breathing is tubular, and the vocal resonance increased. Over the area of deep tenderness in sixth, seventh, and eighth spaces, coarse friction is audible.

Elsewhere lungs are normal.

Pulse 88, and irregular in rate and force. The pulse is small, but hard.

Apex beat in fifth space and nipple line. Epigastric pulsation. No thrill. The first sound at the apex is sharp, and the second re-duplicated. Pulmonary second sound accentuated. No murmur heard at first.

Liver and spleen not felt.

Urine showed a faint cloud of albumen.

January 20.—She began to spit up red or purple-coloured sputum.

January 22.—All deep tenderness has gone; but friction was occasionally heard over the same area.

January 23.—She began to complain of pain in the *left* axilla, situated in the seventh and eighth spaces in the mid-axilla.

There is exceedingly well marked deep tenderness in the seventh and eighth spaces in the mid-axilla, but no superficial tenderness anywhere.

On the night of January 23, friction appeared over the area in the left axilla.

The diagnosis was made of an infarct in the right lung, followed by a second infarct in the left lung; both reaching the surface, and causing slight pleurisy.

February 20.—She suddenly became hemiplegic, without loss of consciousness.

She then gradually got weaker, and died on March 20.

Post-mortem.—Before the examination was begun, the areas of deep tenderness in the right and left axilla were marked out upon the skin. Dr. Fyffe made a careful dissection, and found two infarcts, as follows. In the right lower lobe was an infarct, a little more than an inch across the base, which had caused thickening of the pleura in its immediate neighbourhood, and this area of thickening lay under the area marked out on the surface. A similar recent infarct, causing a patch of pleurisy, was found in the base of the left lung, and this infarct also lay under the area marked out in the left axilla.

Mitral orifice much stenosed, admitting only one finger. About the edge of the valves were some recent vegetations, and a small erosion. Other valves normal. Hypertrophy and dilatation of left ventricle. No micro-organisms could be found in the valves.

Liver and spleen enlarged.

In the brain an infaret was found, causing softening of part of the internal capsule.

(B) *Referred Pain in Pulmonary Disease.*

The second type of pain met with in diseases of the lungs is that which is usually known as referred pain. It is of no direct localising value, but is situated at certain spots on the chest, or may appear to run round the body at various levels. In these spots we recognise the maximum points of certain areas connected with the central arrangements of the nervous system. Pains of this type are usually described as "intercostal neuralgia," "myalgia," "intercostal rheumatism"—names which are either meaningless, or else imply a false pathology. For these pains do not follow the course of the intercostal nerves, but occupy part of the territory of one nerve, and part of that of another. They are said to be "stabbing," "catching," "aching," or "tight;" and in some cases the patient complains of a true girdle sensation, with a feeling as if his chest were tied in at a certain level. The pain is not usually increased, to any marked extent, by each act of inspiration, but is markedly increased by anything that throws increased work upon the diseased lung. Thus the dyspnœa produced by going up a flight of stairs, will frequently bring on, or increase, referred pain, although each respiratory act is not peculiarly associated with an increase of pain.

Such referred pains are associated with tenderness of the superficial structures of the chest. Now it has been long known that phthisical patients are subject to soreness and tenderness of the chest walls, but this tenderness has been considered of little importance. Walshe[1] noticed this superficial tenderness, and points out that "firm pressure over a broad surface gives relief." Valleix[2] pointed out that what he called intercostal neuralgia, was accompanied by three tender points: one behind, one in the axilla, and one on the front of the chest. This observation, Walshe confirms; and

[1] Third Edition, p. 192. [2] "Traité des Névralgies."

points out that no one intercostal nerve is affected, "the pain usually involves the nerves from the sixth to the ninth —in rare instances a single trunk only."

But, in spite of the common neglect of superficial tenderness by the physician, this feature of the disease is well recognised by many patients. They complain that the neck, chest, back, or upper abdomen feels sore and tender, as if it had been beaten or was bruised. They tell you that they have been obliged to loosen their clothes, to wear soft things next the skin; or, in the case of women, that they have been obliged to loosen or remove their corsets. On picking up the superficial structures between the finger and thumb, areas are found, within which the patient complains that the manipulation, however gentle, causes pain. By using the blunt head of a pin these areas can be defined; for the patient either complains that certain parts are sore when touched with the head of the pin, or else he experiences a sensation as if he were being pricked. I must, however, here warn the observer of the fallacies which are attached to the use of the head of the pin upon the anterior and lateral aspects of the chest. He must be very careful, in these regions, that the head of the pin does not impinge on the edge of a rib; for this will cause pain even in a normal individual. Thus I always prefer to lift the skin between the finger and thumb when testing areas on the chest, excepting over such parts, at the breasts,[1] where the pin is obligatory. Here, however, the ribs are so well covered that fallacious results are less likely to be obtained with the head of a pin.

Another fact which shows how purely such pain and tenderness depend upon impulses passing into the central nervous system, is the association of such referred pain on the chest, with areas of referred pain and tenderness on the scalp. This association takes place according to definite rules, which I have laid down in Part II.[2] of this investigation;

[1] In multiparæ the superficial structures of the breast are comparatively insensitive; and superficial tenderness that is well marked over the back and rest of the chest may frequently be almost absent over the breasts, although the breast lies within the tender areas.

[2] BRAIN, 1894, p. 436.

and the occurrence of such scalp tenderness in diseases of the lungs will be repeatedly alluded to in subsequent sections of this chapter.

§ 2. *Conditions necessary to the production of referred pain in diseases of the lungs.*

Referred pain is produced by distention of a sensitive organ from within, or by the exertion upon it of some tearing or rending strain from without. Thus, if the intestine is distended, referred pain may result. If, on the other hand, it is cut with a sharp knife, as in the second stage of inguinal colotomy, no pain is produced; but if, in the process, it is torn or pulled upon, the patient complains of pain. Moreover, it is essential that, at any rate, some of the end organs within the diseased area should be intact, in order that referred pain may result. Thus no true referred pain and superficial tenderness result from a tooth in which the pulp is dead, although much local pain may be caused by inflammation around the root. Thus any disease which prevents all movement in the diseased organ, or any disease that rapidly destroys its tissues, including the nerve endings, will be unaccompanied by referred pain.

Now, in acute lobar pneumonia, the essential process of the disease is a consolidation of one or more lobes of the lung, with complete obliteration of the alveoli by exudation. The air no longer enters the alveoli of the whole consolidated lobe. But, in spite of its profoundly diseased condition, such a consolidated lobe gives rise to no referred pain, for the whole of the end organs are removed from the influence of pressure from within, or traction from without, by this consolidation. Thus the large majority of cases of acute lobar pneumonia exhibit no referred pain or superficial tenderness, and associated with this absence of referred pain there is no referred headache, or tenderness of the scalp. But in a considerable number of cases of acute lobar pneumonia, the consolidation of the lung is associated with pleurisy of greater or less extent. To this is due the local pain, frequently accompanied by tenderness on percussion or pressure, which is not

an uncommon feature of this disease. I give an instance in which both lower lobes were consolidated; but the only pain that was present was localised over the left base, where marked friction was audible.

Case 62.—*To illustrate the absence of referred pain and superficial tenderness in Acute Lobar Pneumonia, and the dependence of the pain frequently present in this disease on coincident pleurisy.* Luke L. (V.P.H., Dr. Thorowgood); aged 38; Wood carver.

On the evening of March 29, 1894, he was seized with cold shivers. The next day pain came on in the left side.

On admission (April 3).—Well-built man. Face of a pinkish-yellow colour, with a patch of herpes about the right, lower lip, extending outwards to the cheek. No marked cyanosis.

Respiration 24. Sterno-mastoids acting slightly. Some subjective dyspnœa. He can lie on both sides, but prefers to lie on the left side. Short paroxysms of cough. At infrequent intervals he spits up a small quantity of greenish-brown, frothy expectoration.

He complains of pain over the middle of the left axilla in the seventh, eighth, and ninth interspaces. No superficial tenderness, but extremely well-marked tenderness on percussion, or deep pressure was evoked in the sixth, seventh, eighth, and ninth spaces from about an inch from the costal margin below to the line of the latissimus dorsi above.

No headache. No scalp tenderness.

The percussion note in the left axilla, and below the level of the sixth spine on the left side, is absolutely dull and resistant. Over this area the breath sounds and vocal resonance are almost completely abolished, and in the sixth, seventh, eighth and ninth spaces in the axilla, marked friction is audible.

The percussion note over the right base and in the right axilla is tubular. Over this area the heart sounds are weak, and the vocal resonance fair. Numerous fine crepitations are heard, but no friction is audible.

The temperature was 102·8° F. (39·3° C.) The pulse was good. The urine contained no albumen.

All pain and deep tenderness rapidly disappeared, and on May 6 the temperature was absolutely normal; and the lungs presented no abnormal signs, except a very slight diminution of note, with weak breathing and vocal resonance below the angle of the left scapula.

Thus the form assumed by the disease is all important in determining the presence or absence of pain in the course of phthisis and other diseases of the lungs.

Three stages—invasion, consolidation, and excavation

—are usually described for tubercular phthisis; but experience shows that they occupy a very variable amount of the field in the clinical picture of any one case. Thus, in the one extreme type, the patient scarcely complains of anything beyond a cough in the mornings, and a feeling that he is not up to the mark. And yet, when he seeks advice, marked signs of consolidation, and, perhaps, of excavation, are present over the upper part of one lung. The temperature may be very irregular—greatly raised one night and scarcely raised at all the next. The patient may gain weight under treatment, and take his food well; but in spite of this, the cavitation may steadily advance. For whilst, at first, the signs of a cavity may extend to the level of the third dorsal spine, in two or three months similar signs may be present as low as the sixth spine. Yet during this time, all referred pain, referred headache, tenderness of the scalp, and superficial structures of the chest may be entirely absent.

From this point onwards the course followed by the case may be very variable. A sharp rub may make its appearance about the angle of the scapula, or in the axilla; and the whole base of the lung may become adherent; or the upper lobe, or the apex of the lower lobe of the opposite lung, may become rapidly excavated. But neither of these processes is associated with referred pain or superficial tenderness. Thus, in an extreme case of this type, the patient may advance far with the disease without suffering from any referred pain.

Moreover, when excavation, or pleurisy, or an association of both processes, form the main feature in the course of a case of phthisis, the parts which are not visibly affected by tubercle tend to become markedly fibrotic. Thus, in such a case, the adherent, but otherwise unaffected, bases are found, *post-mortem*, to contain excess of connective tissue. They are thus in an unfavourable condition to give rise to referred pain when actually invaded by tubercle.

Thus, what may be called the excavation and pleuritic type of phthisis may run on to the end without showing any referred pain or superficial tenderness.

Case 63.—*To illustrate the absence of referred pain and superficial tenderness in those cases of phthisis where excavation is a marked and early feature of the disease.*—Herbert K. (V.P.H., Dr. Heron); aged 26; groom.

He was in splendid health until October, 1893. He was then seized with some acute illness. On the second and third days he lay in bed. On the fourth day he got up, and on the fifth day a cough came on, and has not left him since.

He felt languid and ill, but was able to continue work till November. At the end of November he began to sweat at night, and about the beginning of December he began to spit up with his cough. He has lost 7½ lbs. in four months. He has been free from pain throughout.

On admission (January 22, 1894.)—Well-built man of 26. Not anæmic. No marked cyanosis. Sweats at night.

Respiration 28. Short of breath on considerable exertion, but not when walking quietly. Cough, single; not very troublesome. Small quantity of expectoration, containing tubercle bacilli.

No pain. No headache. No superficial tenderness anywhere.

The right apex moves less than the left, both in front and behind. The percussion note is diminished on the right side, over the first two spaces in front, and as low as the level of the fourth spine behind. Over this area, both in front and behind, marked cavernous breathing and whispered pectoriloquy are heard, with many metallic râles. A few scattered râles are heard behind over the upper part of the right lung; but the breath sounds at the bases and at the left apex are normal. Larynx, normal. No abnormal signs in heart. Tongue, moist. Appetite, poor. No nausea or vomiting. Bowels, normal. Urine contains no albumen, and is acid.

The temperature rose to 102° F. (39° C.) every night, but fell to normal in the morning.

The signs of cavitation at the right upper part of the right lung spread downwards rapidly, and by March 3 cavernous breathing and whispered pectoriloquy could be heard as low as the level of the sixth spine.

Then sharp, dry friction appeared at the angle of the right scapula, and by April 12 the movement of the right base was much impaired, the note was diminished, and the breath sounds very weak all over this base.

The temperature still remained very high, and he had lost 6½ lbs.

No referred pain, headache, or superficial tenderness appeared throughout the whole time he was under observation.

This case is an extreme instance of phthisis, running a course unattended by referred pain. But the disease may advance in a manner that is so different to the instance I

have just given as to form an almost exactly opposite type. When the patient first seeks advice, there is, perhaps, a little deficiency of movement at one apex. The percussion note is not definitely altered in front or behind, but a few sharp crepitant râles are heard over the upper part of the lung on the side where the movement is deficient. There is a good deal of cough, and some expectoration, in which tubercle bacilli can be found, generally in considerable numbers. The patient may complain of some aching pain about the clavicle, but there may be little or no superficial tenderness. Then the signs rapidly increase, and fine crackling râles may become audible over the upper part of the lung as low as the level of the fourth or fifth dorsal spine, and a few râles and rhonchi may be heard creeping round the vertebral border of the scapula. The breath sounds over the upper part of the affected lung are feeble, and the vocal resonance perhaps a little increased. The patient now complains of referred pain in the affected side of the chest, and more or less superficial tenderness is present both on the chest and on the scalp.

Let us suppose that the left side is first affected. This side may now remain quiet for a time, and, although the lower part of the interscapular fossa may be free from râles, they persist over the upper lobe and apex of the lower lobe of the left lung. All referred pain and superficial tenderness may die away. Then, after a varying interval, a sudden outburst of râles and rhonchi may appear over the right side, extending to the level of the angle of the scapula. Referred pain will now reappear mainly over the right side of the chest and back, accompanied by widespread superficial tenderness, more marked on the right side than the left, but present on both sides of the chest.

The acute signs on the right side may now die away, leaving the upper lobe and apex of the lower lobe damaged, as shown by the weak breath sounds and a few irregular râles. The referred pain and superficial tenderness may either disappear, or be very much diminished in intensity and extent.

The next acute outburst may occur on the same or on

the opposite side, and be accompanied by a further development of referred pain and superficial tenderness of the chest and scalp.

Each such acute outburst subsides, but leaves the lung more damaged than before, and, sooner or later, signs of excavation make their appearance, generally at the upper part of the lung first affected. But for some time after the first signs of excavation have appeared, the progress of the case is characterised by periodic outbursts of râles and rhonchi over the lower portions of the lungs.

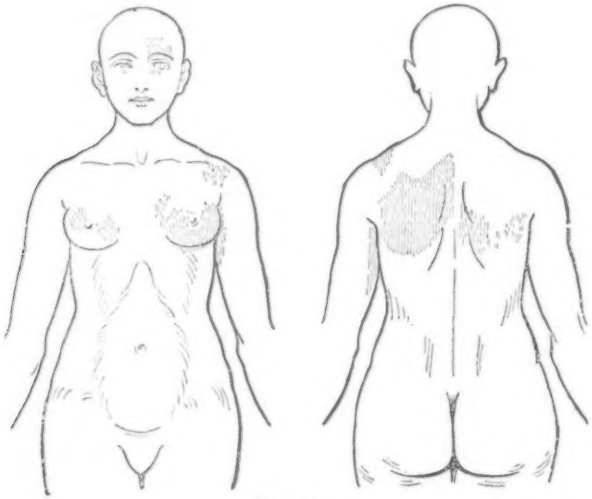


FIG. 15.

To show the superficial tenderness present in a case of phthisis [No. 64] during the implication of the left lung [Jan. 25].

It is this type of phthisis (of which I have given an extreme example in Case No. 64) that is accompanied by referred pain and superficial tenderness in its more exquisite form. The reason for this, I conceive, to be that, during the bronchitic type of invasion, the alveoli are rather encroached upon than primarily affected. The end organs are irritated but not destroyed, and are capable of conveying impressions even from the diseased portions of the lungs.

Case 64.—*To illustrate the type of Phthisis in which the advance takes place by repeated so-called bronchitic attacks.* Catherine C. (V.P.H., Dr. Harris), aged 24; housewife.

About August 1893, she began to have a cough and to spit phlegm, occasionally streaked with blood. She then began to lose flesh. November, 1893, she began to have pain in her left side.

On Admission (December, 15, 1893).—She is a largely-built woman, distinctly wasted. Face pale. No cyanosis. Sweats profusely at night. The temperature was 102° F. (38·9° C.) on the first night. This fell to 98° F. (36·7° C.), in the morning; rising to 100·4° F. (38° C.) the second evening.

Respiration 28. The alæ nasi dilate. Some subjective dyspnœa. Cough not very troublesome. Expectoration, in considerable quantity, consists of small masses of muco-purulent material, streaked with blood, and contains tubercle bacilli.

No pain. No headache. No superficial tenderness.

The movement at the apices is somewhat poor, and the right base moves rather more freely than the left. There is no very marked difference in the percussion note over the apices, but the note over the left apex in front is perhaps a little less resonant than over the right. On the left side, in the first and second interspaces in front, and as low as the second spine behind, the breath sounds are weak, vocal resonance increased, and numerous crackling râles are audible. Otherwise there are no markedly abnormal signs in the lungs.

Pulse 94. Small stroke and regular. Heart sounds normal. Tongue moist, and a little grey; papillæ prominent, not actually furred. Appetite good. Bowels irregular. No nausea. No vomiting. No pain after food. Liver and spleen not felt. Urine acid; no albumen, nor sugar.

January 14.—The temperature ranged to 101° F. (38·3° C.) every night during the first month. She has had very slight referred pains, but now no definite superficial tenderness could be found.

The signs at the left apex have increased. There is no distinct diminution of note over the upper two spaces on the left side in front. Over this area the breath sounds are feeble, expiration distinctly prolonged, and vocal resonance is markedly increased. Numerous fine crackling râles are heard as low as the third rib.

Behind, on the left side, the note is diminished as low as the level of the fourth spine. The breath sounds are exceedingly feeble, and the vocal resonance is a little increased. Fine crackling râles are heard to the level of the fourth spine behind.

No abnormal signs on the right side.

January 25.—She complains of shooting pains in the left side of the chest and left half of forehead, accompanied by superficial tenderness, as in fig. 15.

The cough is not more troublesome, nor the expectoration more profuse. The crackling râles at the upper part of the left

lung are more numerous, and extend down to the level of the sixth spine behind. When the left hand is placed on the right shoulder, these crackling râles are heard along the vertebral border of the scapula, but when the arm is dropped to the side they are not heard at the angle of the scapula.

January 30.—The signs on the left side of the chest have extended a little lower, but have not materially altered. A sudden outburst has, however, taken place on the *right* side, and crackling

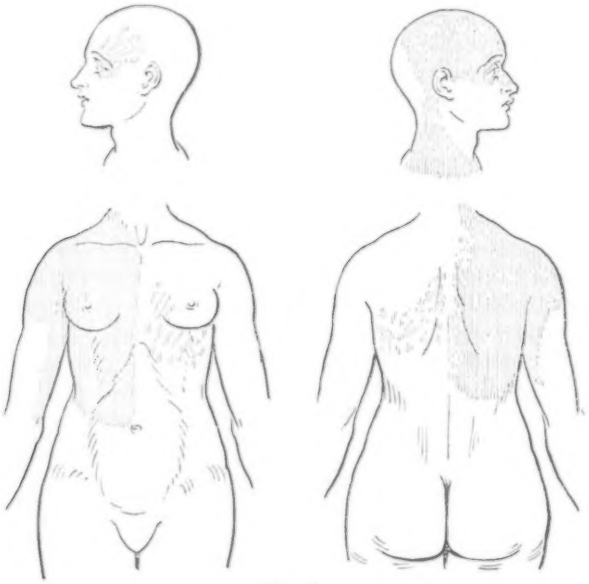


FIG. 16.

To show the superficial tenderness present in the same case of phthisis [No. 64], in a later stage, during the implication of the right lung [Jan. 30].

râles are heard over the whole upper part of the inter-scapular fossa, and about the angle of the right scapula.

The pain has shifted. She complains now of much pain all down the right side, and over the right half of the head. Extremely widespread tenderness is present over the right side of the body and scalp, such as not infrequently accompanies an acute outburst of this kind. (Fig. 16).

February 15.—She is now complaining of pain over the epigastrium, lower ribs, and under the shoulder blades behind. The

headache is mainly on the top of her head. Marked superficial tenderness as in fig. 17.

The appetite is poor. The tongue is clean. Bowels opened daily. No diarrhœa or constipation now. She is beginning to feel a little sick, but has not vomited.

On the left side in front, the dulness extends to the fifth rib. Cavernous breathing and whispered pectoriloquy are heard in the upper two spaces. Behind the second space the heart sounds are very weak, and the vocal resonance increased. Numerous râles audible.

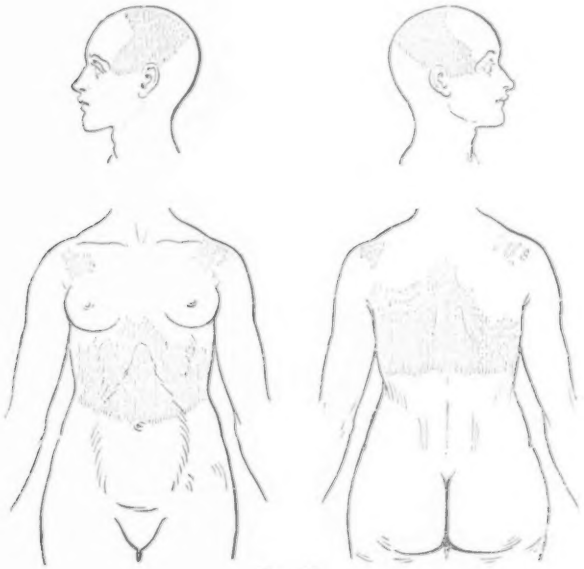


FIG. 17.

To show the superficial tenderness present during a later stage of the same case [No. 61].

Over the upper part of the left lung behind, the note is dull as low as the sixth space. The breath sounds over the apex are very weak, but expiration is hollow, and bronchophony is heard. Behind this to the base the breath sounds are very feeble, expiration is prolonged, and vocal resonance is increased. Râles are heard behind from the apex to the level of the eighth spine.

On the right side in front, râles are heard to the level of the fifth spine. Behind the note is somewhat diminished, but less so than on the left side. The breath sounds are feeble, and

expiration is prolonged as low as the angle of the scapula, and over the whole of this area, vocal resonance is increased, and numerous râles are heard.

Thus both bases are now being steadily affected.

She remained in this stage till March 11, when another outburst took place over the whole right lower lobe, especially about the angle of the right scapula, and in the right axilla.

This produced another widespread outburst of superficial tenderness of both sides of the body and scalp.

The temperature now rose to 103° F. (39·5° C.) every night, and she became very weak.

Thus, in the space of three months, the march of the disease was as follows:—Firstly, affection of the left apex, not associated with referred pain. Secondly, spread along the upper border of the left lower lobe (fig. 15). Thirdly, affection of the apex of the right lower lobe, together with an acute outburst within this lobe (fig. 16). Fourthly, steady advance down both sides within the lower lobe (fig. 17). Fifthly, an acute outburst over the whole of the right lower lobe, from which it never even partially recovered.

Between these two extreme types of invasion and progress innumerable variations occur, but the more nearly the course of a case of phthisis approaches the bronchitic type, the more certainly will referred pain and superficial tenderness tend to appear, and form a feature in the clinical picture. The more nearly it approaches the excavation and pleuritic type, the less likely are referred pain and superficial tenderness to make their appearance.

Thus, when considering the origin of referred pain in any particular case of phthisis, many different factors have to be taken into account. No referred pain accompanies rapid and absolute consolidation of one lobe, or its subsequent excavation. On the other hand, partial implication of the lung by multiple foci of disease [not acute miliary tubercles] scattered amongst relatively healthy tissue, or the gradual opening out of a collapsed or airless patch, are particularly liable to be associated with referred pain. Thus, the physical signs which most frequently stand in connection with the development of referred pain in the course of a case of phthisis, are small sharp, dry râles and sibilant rhonchi. But these small râles may be present for months over some particular area of the chest without there being any referred pain. Thus, it is not the quality nor the nature of the

adventitious sounds which is important, but the sudden appearance of these small râles and sibilant rhonchi over parts where they have not been audible before. The larger and moister râles are not usually associated with referred pain.

§ 3. *An attempt to connect the presence of certain areas of superficial tenderness with disease of certain portions of the lung.*

Now that we have considered the conditions and physical manifestations, which are associated with referred pain in the course of a case of phthisis, it remains to be seen how far such referred pain represents the position of the disease.

At the outset, we are met by the difficulty that every referred pain, when sufficiently intense, or of sufficiently long duration, passes over and affects the same areas on the opposite side of the body. Thus, if the patient complains of pain under the shoulder blades and across the lower part of the chest, we have to enquire on which side the pain began, and on which side it is worse. Both, however, depend upon the patient's memory and observation, and the answers are, therefore, of but little use in practice. Far more valuable is an examination of the condition of the superficial tenderness on the two sides of the body. If the tenderness on the one side is very marked, and extends completely round the body from the middle line behind to the middle line in front; whilst on the opposite side the intensity is less, and the distribution spotty, and only the maximum points well marked, the referred pain probably began over the former and spread to the latter side. Sometimes the headache and scalp tenderness afford a guide to the side upon which the disturbance began, for the headache and scalp tenderness are usually more marked upon that side upon which the pain on the body is most marked.

By intensity of tenderness, I do not mean an expression of the patient's opinion as to which side is the most tender. I judge of the intensity of the tenderness (1) by the sensation produced by the blunt head of a pin. Stimulation with the

blunt head of a pin may only cause a feeling of soreness over the tender area, if the tenderness is but slight, or, if the tenderness is more acute, the patient may complain that he is being pricked or stabbed. Thus, in the minor degree of tenderness, the patient recognises a change in the sensibility of his skin, whilst in the major degree he complains of a change in the nature of the stimulus. (2) By the completeness with which the bands of tenderness are represented on the surface of the chest. (3) By the exaggeration of the superficial reflexes.

But another disturbing factor in the attempt to determine the localising value of referred pain in phthisis lies in the fact that a fresh outbreak in one part of the lung is usually associated with a rise of temperature. Now, wherever referred pain and tenderness are present, and a sudden rise of temperature occurs, the pain, and particularly the tenderness, tend to spread widely. Thus a sudden acute outburst over the upper part of one lower lobe of the lung will not unfrequently lead to exceedingly widespread tenderness of the neck, trunk, and scalp, mainly on the side of the fresh lesion, but also on the opposite side. But though a rise of temperature tends to cause spreading in those cases where referred pain and tenderness are already present, it does not tend to generate tenderness in those cases where it would not otherwise appear. Thus where the lesion consists of a large cavity, the temperature is frequently raised, and even a rigor may occur; but no referred pain or superficial tenderness of the body or scalp appear.

A third difficulty that meets us is the tendency to spreading, and the consequent diminution in the localising value of the referred pain, produced by the cachexia which accompanies the disease. Any referred pain and tenderness which occurs in phthisis tends to spread, owing to the deficient resistance within the nervous system, produced by the phthisical cachexia. Thus the referred pain and superficial tenderness produced by the inflammation of the pulp of a tooth will tend to spread beyond the limits usual when that particular tooth is affected. Hypermetropia headaches

also tend to become much more troublesome, and the tenderness to spread beyond the forehead, in consequence of the patient's cachectic condition,

Case 65.—*To illustrate the tendency to spread shown by all referred pain, of whatever origin, owing to the cachexia of phthisis.* Sarah Ann T. (V.P.H., Dr. Sainsbury).

Within the last six months she has had cold after cold, and repeatadly lost her voice. She has grown much thinner, and sweated at nights throughout this period. Slight hæmoptysis a month before admission.

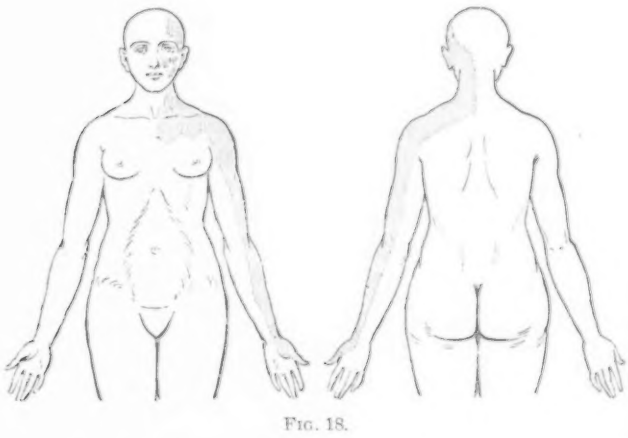


FIG. 18.

To show the wide-spread tenderness produced by disease of the pulp of the second lower molar [Case No. 65]. The great extent of this tenderness is probably due to the cachexia produced by phthisis.

I will not enter into great detail, for this case is quoted to show how any visceral pain tends to spread in phthisis whatever may be its origin.

Dulness was present over the right side behind to the level of the sixth spine. Bronchial breathing and pectoriloquy were heard over the upper part of this area, and a considerable number of râles were audible. There was a small ulcer on the left vocal cord.

She remained free from pain till five weeks after admission, when she developed pain in the second lower molar on the left side. The pulp cavity was exposed. Neuralgia came on at the same time in the face, with pain in the left ear. Then all the

teeth in the left half of the lower jaw up to the canine began to ache. The whole left side of the face, neck, and a part of the left arm began to ache. She feels as if all the power of lifting her left arm were gone, but she can use her fingers perfectly.

There is extensively widespread superficial tenderness over the left side of her face, neck, and radial side of left arm. (Fig. 18).

The temperature was 100·2° F. (37·9° C.).

The whole disappeared within four days of removing the second lower molar tooth.

Now such a widespread disturbance is not usual,[1] except where the patient is anæmic and cachectic. This case shows how the cachexia of phthisis leads to a condition in which referred pain spreads with great rapidity.

Bearing in mind these sources of fallacy, the referred pain and superficial tenderness appear on the same side of the body and scalp as the lesion in the lung.

An attempt to further differentiate the referred pain and superficial tenderness which occur in phthisis is met by a difficulty in nomenclature. To say that certain referred pain is due to an affection of the upper lobe, and pain in another position is associated with affection of the lower lobes would be simple enough. But the upper lobe of one lung is rarely affected apart from the apex of the lower lobe, and thus a statement in this form would be both unpractical and indefinite. Again, from the point of view of the advance of the disease, the lower lobe is made up of several portions. For, as Fowler[2] showed in his admirable paper, the apex of the lower lobe is easily affected in the course of tuberculous phthisis, and the disease then progresses along the upper and outer portion of the lower lobe beneath the interlobular septum. Spreading later to the other portions of the lower lobe of the lung.

Again, the affection of the lower lobe is more often the cause of referred pain than the upper lobe. For the ordinary process of destruction in the upper lobe is one of consolidation followed by excavation. But the mode of extension towards the base is not usually by an advancing line of

[1] *Vide* part ii. of this investigation, BRAIN, 1894, p. 414, and p. 361, fig. 10.

[2] *The Localisation of the Lesions of Phthisis.* Kingston Fowler. London, 1888.

consolidation, but by scattered nodules of infiltration often arranged in a racemose manner. Now this is just the condition which is likely to lead to referred pain and superficial tenderness, and thus the implication of the lower lobes is far more commonly the cause of referred pain in phthisis than that of the upper lobes.

Another difficulty that meets us at the outset is familiar to everyone who has performed many *post-mortem* examinations. The amount of the lesion discovered after death is frequently considerably in excess of the signs during life. Thus all the following attempts to show that certain portions of the lung are associated with certain segments of the central nervous system must be accepted with a liberal margin for error. Such an attempt at differentiation would be quite impossible if chronic phthisis advanced steadily down the lung. It is only rendered practicable by the fact that it is usually the small acute outbursts in the course of the chronic disease that cause referred pain and superficial tenderness, and that the signs audible during such an outburst, more nearly represent the extent of the disease than the chronic signs present between any two such outbursts of fresh activity.

Purely for the purpose of attempting to associate the extent of the pulmonary lesion in phthisis, with the areas of superficial tenderness on the chest, I have been led to divide the lung into the following parts. I do not suppose these divisions have any permanent value, they are purely rough empirical divisions, by which the commoner lines of spread may be classified for association with areas of referred pain and tenderness in the one disease, *i.e.*, phthisis. These divisions are marked out as follows :—

(1) Place the hand upon the opposite shoulder,[1] so that the angle of the scapula becomes tilted upwards and outwards. Draw a line from the fifth dorsal spine (sixth dorsal vertebra) outwards and slightly downwards to the angle of the tilted scapula. Above this line lies the upper part of the lower lobe.

(2) Let the hand fall to the side. Then draw a line from

[1] *Cf.* Fowler, *op. cit.*

the seventh dorsal spine outwards and slightly downwards following the line of the eighth rib, and passing below the angle of the scapula. Above this rough line lies the middle division of the lower lobe.

(3) From this line to the base of the lung lies the third division of the lower lobe.

The upper lobe of the lung is but rarely affected without some implication of the apex of the lower lobe. But in those few cases where the disease was very early, and the râles were mainly present in front, and in the supra-clavicular fossa, the pain and superficial tenderness lay mainly within the third and fourth cervical and third dorsal areas. Now, early phthisis is usually said to cause "pain over the lung," or "tenderness over the upper intercostal spaces." But where this pain and tenderness are present, a careful examination shows that the pain is frequently referred and not local, and that the tenderness is superficial. Thus, the tenderness frequently runs up the back of the neck, and is present over parts of the neck under which no lung tissue is situated. In the same way, the little tag of tenderness on the arm belonging to the third dorsal area can frequently be demonstrated at a distance from the lung.

As soon as acute signs become audible in the neighbourhood of the fourth and fifth dorsal spines, and the râles are found creeping round the vertebral border of the scapula when the hand is placed on the opposite shoulder, the third, fourth, and fifth dorsal areas tend to make their appearance.

When the disease attacks the middle division of the lower lobe, and the râles are found clustering about the angle of the scapula (with the arm by the side) and extend into the lower part of the axilla, the fifth, sixth and seventh, but particularly the sixth dorsal areas, make their appearance.

When the lowest division of the base of the lung becomes affected, the tenderness lies mainly over the seventh, eighth, and ninth dorsal areas, but most commonly over the seventh and eighth in phthisis.

I have entirely neglected the middle lobe of the right lung, because it is almost impossible to localise the disease

affecting it, even with the roughness that is alone possible elsewhere. Moreover, it is frequently found, *post mortem*, to have escaped, although the greater part of the right lung is affected.

The lung is, therefore, associated with the third and fourth cervical, and the third to the ninth dorsal areas inclusive. The second dorsal escapes, as a rule, except in such cases where we have reason to suspect that the tenderness has spread. Sometimes, however, it seems to make its appearance even where such spread seems improbable. The possibility of variation must always be borne in mind.

In my first paper, I did not place the supply of the lung lower than the fifth dorsal segment; for from this point downwards we reach the gastric areas, and at that stage of the research, I did not feel justified in associating any pain and tenderness accompanied by gastric disturbances with lesion in the lung. My present opinion on this matter is embodied in §4 of this chapter.

As lesions of the upper part of the lung are associated with pain and tenderness within the third and fourth cervical and upper dorsal areas, they are accompanied by superficial tenderness over the forehead and around the eyes. On the other hand, as soon as the lower lobes are considerably affected, the scalp tenderness will travel back to the temporal, vertical, or even to the parietal regions. The lower the lesion in the lung, the nearer the tenderness over the body approaches the ninth dorsal area, and the further back is the tenderness on the scalp.

To summarise the conclusions to which we have arrived in this section:—

(1) All referred pain and superficial tenderness which makes its appearance, from whatever cause, in the course of subacute phthisis tends to spread widely. This is probably due to (A) the cachexia, and (B) the temperature which accompanies the disease.

(2) Referred pain produced by the disease of the lung was most marked, and of greatest intensity on the same side of the trunk as the fresh lesion. Both pain and tenderness may, however, secondarily make their appearance on the

opposite side of the lung, but appear later, disappear earlier, and are not of the same intensity as on the side of the lesion.

(3) The innervation of the lung is connected with the third and fourth cervical segmental areas, and with all the dorsal segmental areas from the third to the ninth. The lower lobe of the lung is particularly connected with the dorsal areas, especially from the fifth dorsal downwards to the ninth.

§ 4. *Gastric Disturbances in the course of Phthisis.*

We have just seen that referred pain and superficial tenderness appear over the middle dorsal areas when the bases of the lungs become affected with disease of a certain type. But these areas, from the sixth to the ninth dorsal, stand in close relation with the stomach, especially those on the left side. Thus two organs, the bases of the lungs and the stomach, send sensory impulses into the same segments of the central nervous system. Now, when any two organs send impulses into the same segments of the central nervous system, an affection of the one which causes referred pain and superficial tenderness, tends to cause a reflex disturbance in the other organ that refers into the same areas. For instance, an inflammation of the pulp of the wisdom tooth of the lower jaw causes a soreness of the fauces and a sense of pain and swelling on the same side as the diseased tooth. Glaucoma will set those teeth aching which refer into the same areas that have become tender owing to the rise of tension in the globe. Thus, it is not surprising that the sudden appearance of referred pain and tenderness over the sixth to the ninth dorsal areas, in consequence of a fresh outburst in the lung, should be accompanied by symptoms referred to the gastro-intestinal tract.

But tubercular phthisis is in itself associated with gastric symptoms, and it thus becomes very difficult to say whether the pain, tenderness, nausea, and vomiting are, in reality due to the fresh affection of the lung, or to some intercurrent gastric attack. In some cases, however, the gastric dis-

turbance seems to be clearly secondary to the fresh outburst in the lung.

The dyspepsia which may occur in the course of tubercular phthisis can be divided clinically into three groups:—

(1) The *prodromal* dyspepsia which occurs during a period of the disease when the signs in the lungs are either absent, or exceedingly slight.

(2) The dyspepsia of *invasion*, which occurs during the gradual invasion of the lung by the tubercular disease.

(3) The dyspepsia of *dissolution*, occurring in the last stages of the disease.

Now, the dyspepsia of dissolution is very rarely accompanied by referred pain and superficial tenderness. It is mainly characterised by flatulence, eructations, and signs of dilatation of the stomach, and feeble motor and sensory activity. Thus, this form of gastric disturbance does not come within the scope of this enquiry, owing to the fact that it does not cause referred pain and superficial tenderness.

The prodromal dyspepsia, on the other hand, is accompanied by marked and widespread referred pain and superficial tenderness. But at the time it occurs the signs in the lungs are either absent, or exceedingly slight; and thus it is certainly not due to what may be called sympathetic reference. It seems to stand in relation with the cachexia which signalises the onset of tubercle of the lungs in the same way that gastralgia so frequently accompanies another general state — anæmia. I do not mean to say that this state is an anæmia, for it differs in certain striking particulars from a true anæmia, and I, therefore, prefer to speak of it as a cachexia. Now there are certain general states, which in themselves seem to be peculiarly associated with, more or less, widespread referred pain and superficial tenderness. To these general states belong the prephthisical cachexia, and certain anæmias, and I shall, therefore, postpone the full consideration of this prodromal dyspepsia, the outcome of the prephthisical cachexia, until I come to treat of these general states. I give the following case (No. 66) as an instance of this form of dyspepsia.

Case 66.—*To illustrate the referred pain and superficial tenderness sometimes associated with the prodromal dyspepsia of phthisis.*—Janet N. (V.P.H., Dr. Sainsbury).

January, 1887.—At the age of 15, she was admitted to V.P.H., complaining of loss of appetite and weakness. She had not begun to menstruate. She was then pale and thin. She vomited her food as soon as she took it, and had much pain at the epigastrium, increased by taking food. The abdominal walls were

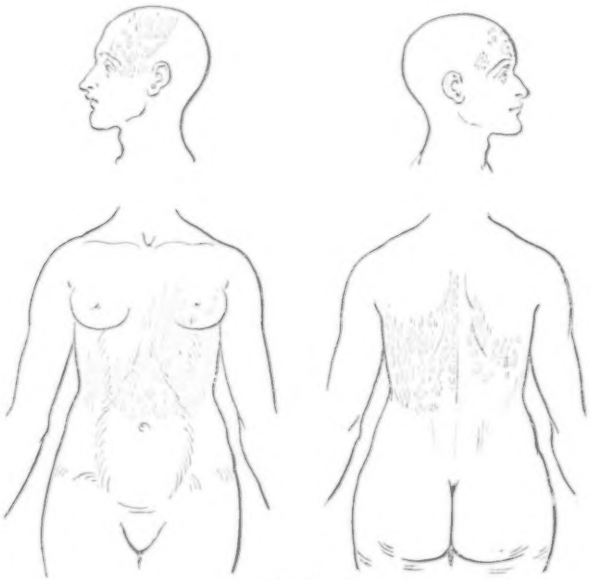


FIG. 19.

To show the superficial tenderness present during the course of the prodromal dyspepsia in Case No. 66.

hyper-æsthetic. Tongue, red and raw looking. Teeth, good. A systolic (hæmic) murmur was heard over the upper part of the sternum; the pulmonary second sound was increased. The temperature was normal throughout.

She left the hospital much improved, but throughout the next four years had several similar attacks.

January, 1891.—At the age of 19 she was again admitted. She had now developed a slight hacking cough, which was only followed by expectoration in the early morning. The sputum contained scarcely any purulent material, and Dr. Hadley was

unable to find any tubercle bacilli. She had been losing flesh for three months, and sweated slightly at night. No definite abnormal signs could be found in the lungs. A systolic hæmic murmur was heard over the second left interspace, and a hum was audible in the veins of the neck. The teeth were still good. Tongue, red. Appetite, bad. Suffered a good deal from flatulence.

February 10.—The tonsils swelled, and the lymphatic glands in the neck became enlarged. The gland in the neck remained enlarged for a considerable time.

She much improved, but in July, 1893, began to feel tired and faint, and to suffer from nausea, whether she took food or not. Vomiting now returned, and she again began to suffer from a slight cough.

October 24, 1893.—She was again admitted to V.P.H. She was now 21, but looked older. The face was pale, of a somewhat opaque white, with a slight flush on each cheek. The ankles swell at the end of the day, but there is no œdema when she is kept in bed. She sweats slightly at night. She was fairly well nourished, and showed no signs of wasting.

She complained of palpitation and shortness of breath. No objective dyspnœa when in bed. Slight single cough, especially in the morning. Expectoration scanty, consisting of a few muco-purulent lumps, in which no tubercle bacilli could be found. She suffers from pain in the epigastrium and back, accompanied by considerable superficial tenderness as in fig. 19. She complains mainly of bitemporal headache, and this headache is accompanied by considerable superficial tenderness of the scalp.

The only abnormal physical signs in the chest were slight diminution of movement over the left apex, with very slight diminution of the percussion note. A few crackling sounds were heard in the upper two spaces on the left side in front. Pulse, 72; low tension. Hæmaglobin, 50 per cent. Red corpuscles, 60 per cent. (3,000,000 in a cub. mm.). No hæmic murmur could now be heard in the chest.

The teeth have decayed very rapidly. The upper central and lateral incisors have gone, and are replaced by a well-made plate. The left second bicuspid, first molar, and right second molar, in upper jaw, exist only as stumps. In addition, many teeth in both jaws show signs of caries.

Tongue, cracked transversely, dry in the centre, and red, but not furred. Appetite, bad. Feels sick at varying times after food. She cannot take fat in any form. Vomiting occasionally. Some flatulence. Bowels opened daily; not constipated.

Liver and spleen cannot be felt.

Menstruation began at 17. Irregular, at intervals of one to two months. She menstruated during her stay in hospital, and suffered from considerable pain and widespread superficial tenderness, which gradually disappeared when the menstruation ceased.

The pain generalised two days before the flow came on. The flow lasted two and a-half days, and the amount was scanty.

The temperature was very irregular. It was invariably raised in the evening, but whereas it sometimes only reached 99° F. (37·2° C.), it occasionally ran up to 100·6° F. (38·1° C.), falling again in the morning to 98° F. (36·7° C.).

During a stay of eight weeks in the hospital she gained 7 lbs. in weight, and much improved.

January, 1894.—The cough returned, and she again became weak and languid, but without vomiting. She had been under continuous treatment with iron, and the hæmaglobin is now 100 per cent., and the red corpuscles 97 per cent. (4,860,000). The movement of the left apex is still somewhat deficient, and a few crackling râles have reappeared both in back and in front on the left side.

August, 1894.—She married. When she became pregnant the vomiting and pain came on again, and she was in bed four months.

August, 1895.—The child was born, and from that time the vomiting and gastric symptoms have much improved, but she began to suffer from extreme weakness. She began to sweat at night again.

February, 1896.—She is obviously thin, and has lost a stone in weight. Sweats profusely at night.

Tongue red and moist. Appetite was very good, but she cannot bear fat of any kind. No pain after food. No headache. No nausea. No vomiting. She now takes large quantities of food, and after meals bitter stuff rises into the mouth, but does not cause her any discomfort. A good deal of flatus at all times.

Coughs, especially in the morning. Expectoration, thick and yellow.

Deficiency of movement at left apex, back and front. Note at left apex not good, but note at right apex, back and front, is considerably diminished. Over upper two spaces, on right side in front, breath sounds are weak, and vocal resonance is increased. Over upper part of right lung behind, breathing is bronchial, vocal resonance is increased, and fine crackling râles can be heard as low as the level of the fifth spine.

Thus the signs of phthisis have now become evident, and the whole character of the clinical picture has altered.

There now remains for consideration, a group of cases of phthisis in which referred pain and tenderness make their appearance over the sixth to the ninth dorsal areas, during the course of the invasion of the lung by the disease. The difficulty in such cases lies in tracking the referred pain and tenderness to its fundamental cause, and, in many

cases, it is practically impossible to say whether the lung or the stomach is the organ that gives birth to the referred pain. However, I think, some help in this difficult task can be gained by dividing this *clinical* group into three *physiological* subdivisions. These three subdivisions of the dyspepsia of invasion I will now consider seriatim.

(*A*) *Pseudo-Gastric Disturbance.*

The patient's general condition may be good, the tongue clean, appetite fair, bowels regular, and referred pain may be absent, superficial tenderness may be absent or only slightly marked, or may, perhaps, be situated entirely over the upper areas of the chest and the front of the scalp. All tenderness over the sixth to the ninth dorsal areas, on the body, and from the fronto-temporal area backwards on the scalp are absent.

Then a slight recrudescence of activity takes place in the lung disease. Fine dry râles, perhaps accompanied by sibilant rhonchus, appear behind over that part of the lung which lies at the level of the seventh dorsal spine. Referred pain accompanied by more or less marked superficial tenderness, appears in one or more of the areas from the sixth to the ninth dorsal, accompanied by scalp tenderness over the temporal, vertical, or parietal areas. The outburst of fresh activity in the lung will probably be accompanied by some rise of temperature, and thus the pain and superficial tenderness, though more marked on the side of the affected lung, will tend to double at the same level of the central nervous system; this is peculiarly liable to happen on the scalp.

But the disturbance of any organ, whether of the head or trunk, which causes well-marked bilateral referred pain and superficial tenderness of the temporal region is associated with nausea, loss of appetite, and possibly, vomiting. Thus, this nausea, and even vomiting, can be excited by affections of the eye, although there is no tenderness over the gastric areas of the trunk.

Thus, when a slight recrudescence of activity occurs in that part of the lung which lies below the angle of the

scapula, the patient may complain, not only of referred pain and tenderness over the gastric areas of the trunk and temporal region of the scalp, but also of loss of appetite and nausea; later, actual vomiting may occur. The tongue is at first quite unaltered, the bowels remain regular, and the evacuations are unchanged in character, the pain is not increased by food, and vomiting does not necessarily relieve the pain and headache.

On the other hand, anything which relieves the pain and headache, will remove the nausea and vomiting. Thus, mustard leaves applied to the maximum spots of the affected areas of the chest or back, or a dose of phenacetin (which acts more quickly on the headache, but also on the referred pain of the trunk) will remove the nausea and vomiting in this mild and purely reflex type of gastric disturbance.

Such a condition rarely lasts long, for, if it does not pass off within forty-eight hours, it tends to merge into the second form next to be considered.

I might have selected a case of ordinary chronic phthisis as my example of this form of gastric disturbance, I have, however, chosen case No. 67, on account of the shortness of the attack of pain and headache, and the complete return of appetite, with the cessation of the nausea and vomiting.

Case 67.—*To illustrate the first, or reflex type, of gastric disturbance which occurs in the course of phthisis.* Ada W. (V.P.H., Dr. Harris), aged 17. Admitted, August 17, 1894.

When a child she had a fever, about which no details can be obtained. Since then she has always been under a doctor for her chest. Her cough has become worse during the last five years.

She is a small girl of 17, but looks much older. She is ill-developed, and the fingers are markedly clubbed. There is no wasting, the body is well covered, and the breasts well formed. She does not sweat at night. There is a marked lateral curvature with the concavity towards the left in the mid-dorsal region.

Respiration 24. No objective or subjective dyspnœa, except when she exerts herself. The dyspnœa then becomes marked. Sometimes she does not cough throughout the day. Then, on first waking, she has a paroxysm of coughing, in which she retches, bringing up a large quantity of greenish-yellow masses of stale smelling muco-pus. Many most careful examinations failed to show any tubercle bacilli.

She is usually free from pain and superficial tenderness.

The whole left side is somewhat retracted, and does not move with respiration. Back and front this side is dull. Over the upper part of the left lung the breath sounds are weak, expiration prolonged, and vocal resonance diminished, but below the level of the sixth spine the breath sounds are almost tubular, and bronchophony is audible. These signs extend to the extreme base. From top to bottom on the left side large moist consonating râles are heard. Thus the disease on the left side seems to be old, and the signs did not alter appreciably in three months.

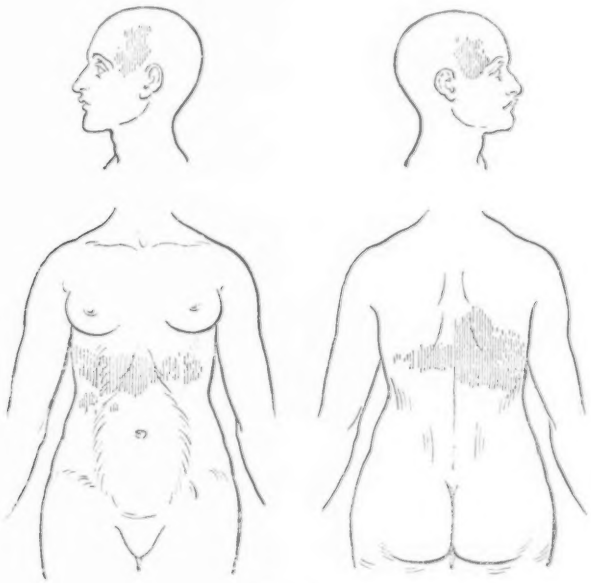


FIG. 20.

To show the areas of tenderness present during an attack of reflex gastric disturbance in Case No. 67.

The right side of the chest is free from abnormal signs, except for a few very large moist râles at the right base.

The pulse was 100, very small, regular, and somewhat hard.

The apex beat seems to be drawn up into the third space on the left side. Heart sounds are normal.

Tongue normal and moist. Appetite good. No pain, nausea, or vomiting (except with the paroxysms of coughing). Bowels normal.

Urine 1015, acid, and containing a considerable quantity of albumen.

She continued in this condition until September 18, when she began to complain of pain under the right shoulder, and in the epigastrium. Some pain on left side, but to a less degree. Bi-temporal headache. This pain was associated with marked superficial tenderness, as in fig. 20, both of body and scalp.

She felt very sick, and vomited twice. The appetite was not good, but she could take her food, and the food caused no pain. The pain was always present whether she took food or not. The tongue was clean and moist. The motions were unaltered.

The cough was not increased, and the sputum still had the stale smell, but was not truly offensive.

At the right base the breath sounds are bronchial, the vocal resonance increased, and a large number of râles have made their appearance below the angle of the right scapula.

The temperature which usually only touched 99° F. (37·4 C.), ran up to 101° F. (38·3° C.)

September 19.—By the evening the pain, tenderness, nausea, and vomiting had disappeared. She took her tea with a good appetite. The temperature did not rise above 99·4° F. (37·5° C.)

She had several similar slight attacks, in the course of the three months she was under observation, apparently due to an infection of the comparatively healthy side by the pus from the dilated bronchi of the left lung, which was in a fibroid condition.

(B)—Sympathetic Hyper-æsthesia of the Stomach.

If the referred pain and tenderness over the gastric areas, produced by the condition of the lung, continues for some length of time, or is of marked intensity from the beginning, the stomach seems to become hyper-æsthetic by sympathy. For not only do nausea and occasional vomiting make their appearance but the pain is distinctly increased by the ingestion of food, and is partly relieved by vomiting. Such patients complain that pain is always more or less present, but that it is markedly increased after taking food and, if vomiting occurs, the pain is relieved, and sinks back to the amount that was present before the food was taken. In this connection it is interesting to notice that if the sixth and seventh dorsal areas are mainly affected, the pain will be increased almost immediately the food is taken into the stomach, and the patient may vomit practically unaltered food within twenty minutes of its ingestion. If, however,

the ninth dorsal area be mainly affected and the upper gastric areas are either absent, or but slightly developed, the pain will not be increased until a considerable time after the food has been taken, and vomiting may be completely absent.

The tongue remains unaltered, as a rule, in this form of gastric disturbance. If it were clean and red, or a little furred, soft and moist it does not alter, at any rate, at first, although the gastric symptoms are very marked. In the same way the action of the bowels and the character of the evacuations are not markedly altered, excepting in consequence of the rejection of food by vomiting, or some alteration in diet prescribed by the physician. Flatulence and the eructation of fluid into the mouth, are not usually symptoms of this condition.

Thus the whole of the symptoms are those of irritability of the stomach without obvious signs of gastric mischief. As soon as the outburst of fresh disease in the base of the lung clears up, or ceases to advance, the referred pain and superficial tenderness over the gastric areas ceases, or diminishes, and with this cessation or diminution of the pain and tenderness the gastric symptoms disappear. The explanation would seem to be that the outbreak in the lung pours pain impulses into those segments of the nervous system which stand in nervous connection with the affected part of the lung. But the stomach also stands in connection with the same segments, and thus becomes hyperæsthetic, in exactly the same way as the testicle may become hyper-æsthetic owing to a renal calculus.[1]

The first case I have chosen as an illustration shows the outburst of referred pain and tenderness, with the implication of the left base. The base of the lung was permanently damaged, but the referred pain and tenderness disappeared when the acute increase in the disease passed away.

The second case shows the disappearance of the gastric symptoms, with the clearing up of an acute outburst at the left base, and their reappearance when the base became again affected.

[1] *Cf.* BRAIN, 1893, p. 78.

Case 68.—*To illustrate the second type of gastric disturbance associated with the invasion stage of phthisis.* (*Sympathetic gastric hyper-æsthesia*). Edward O. (V.P.H., Dr. Thorowgood); aged 34, wire layer for electric light.

In the winter of 1890 had a bad cough and spat much phlegm. Got better, but continued to cough all through the summer. In the winter of 1891 got very bad again, and could not work. He attended Brompton Hospital, and much improved, but in October, 1892, became worse again. Wasted greatly, sweated at night; cough very troublesome. He again improved somewhat, but in October, 1893, got worse than ever.

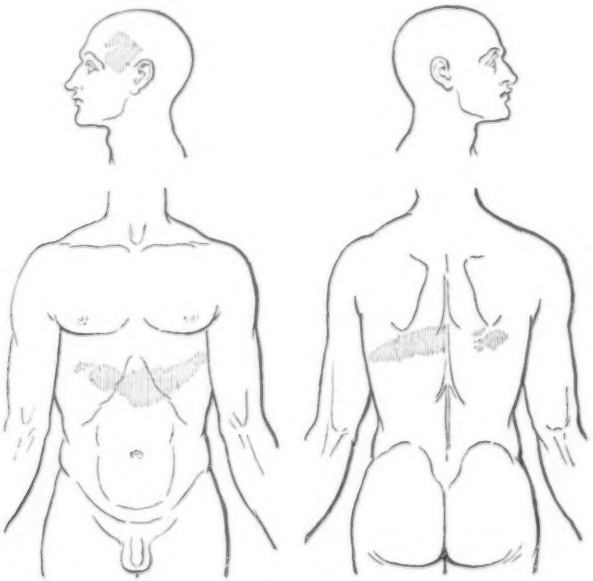


FIG. 21.

To show the areas of tenderness present in Case No. 68 during an attack of sympathetic gastric hyper-æsthesia.

On admission (April 9, 1894).—Small man of 34. Face, cachectic, of a somewhat greasy pallor. Slight pink congestive flush on the cheeks. No cyanosis. Considerably wasted.

Respiration 22. Accessory muscles of respiration acting slightly. Very short of breath, on exertion. Cough troublesome in the morning, but not troublesome in the daytime. Large amount of greenish-yellow, round masses of muco-purulent expectoration containing tubercle bacilli.

He complains of pain in the epigastrium, over the left costal margin, and below the angle of the left scapula. This pain is worse on the left side, than the right. Beautifully marked superficial tenderness. (Fig. 21). Headache over left temple accompanied by well-marked, superficial tenderness.

In Front: movement poor over both apices. Percussion note diminished over the upper three spaces on the left side. Over the left apex the breathing is bronchial, and bronchophony is heard. Over right apex breath sounds are weak, expiration prolonged, and vocal resonance diminished. Many crackling râles all over the front of the chest.

Behind: on the right side the note is dull, to the level of the angle of the scapula; over this area expiration is prolonged, vocal resonance increased and many rhonchi and râles are heard. From the angle of the right scapula to the base the breath sounds exceedingly weak, vocal resonance is much diminished and irregular, crackling sounds are heard. On the left side the percussion note is diminished to the level of the fifth spine. Over this area expiration is prolonged, vocal resonance increased, and many crackling râles and much inspiratory rhonchus is audible. From the level of the fifth spine downwards the breath sounds are harsh, expiration a little prolonged, but vocal resonance is unaltered. Many sharp râles and inspiratory rhonchi are heard over the lower part of the left lung.

Thus the signs in the lungs pointed to an old affection of the right base, and a recent affection of the left base.

Pulse 88, regular. Heart sounds normal.

Appetite very bad. He complains that the pain is increased by food. Feels sick, and retches within half-an-hour of taking food. Has vomited several times with this retching, apart from coughing. The vomit consists of almost unaltered food. Tongue small, moist, a little tooth indented. Bowels opened daily without drugs. Motions normal.

Liver and spleen not enlarged. Urine normal. Temperature rose every night to 100° F. (37·8° C.) falling in the morning to normal.

By April 20 all pain, headache and superficial tenderness had disappeared. He no longer felt sick after food. Food caused no pain. Appetite good.

The signs in the lungs on the right side remain the same. Over the upper part of the left lung behind, to the level of the fifth spine, the dulness is unaltered, and breath sounds and adventitious signs remain as before. Below this point all over the left base the breath sounds are good, vocal resonance normal, and absolutely no râles or rhonchi can be heard, or have been audible for several days.

On May 9. An exactly similar attack of nausea, vomiting, pain after food began, associated with another outburst at the left base. He left the hospital with persistent signs below the angle of the left scapula.

Case 69.—George R. (V.P.H., Dr. Sainsbury); aged 39, cellarman.

He was quite well till 1891, when he had an attack which was called "Influenza." He remained in bed for two weeks, and for the next three weeks was troubled with cough and spitting. July, 1892, began to suffer with pain in the left side and cough returned. January, 1893, had another attack of "Influenza" with pain in the left shoulder. He coughed, sweated at night, and lost much flesh. The cough has continued ever since. April, 1893, began to spit blood and the expectoration continued to be blood-stained for two weeks.

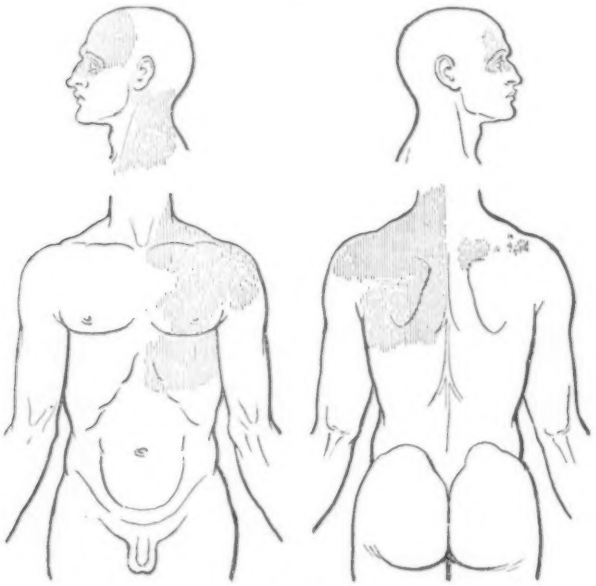


FIG. 22.

To show the areas of superficial tenderness in Case No. 69, produced by the invasion of the base of the left lung. This tenderness was associated with marked gastric disturbance.

Owing to the nature of his employment he takes alcohol somewhat in excess; five or six pints of beer daily.

On admission (October 16, 1893). Large well-built man, evidently somewhat wasted. Says he has lost 14 lbs. in six months. Face of a yellowish pallor. No flush in cheeks. No cyanosis. No profound anæmia.

Respiration 20. Accessory muscles quiet. Dyspnœa on exertion. Hard hacking cough. Spits up a considerable quantity of small muco-purulent yellowish masses tinged with bright or dark red blood.

Some pain in left supra-clavicular fossa, and over the posterior aspect of the left shoulder joint. Distinct superficial tenderness over the spots to which the pain is referred.

In front: right apex moves somewhat better than the left. Percussion note diminished in first two spaces on left side. Expiration is prolonged and vocal resonance increased as low as third space on left side. Fine dry râles heard over the same area. Over the right apex breath sounds are fair, vocal resonance a little increased, and a few scattered râles are heard.

Behind: on the left side the dulness extends to the level of the sixth spine. Expiration is prolonged and vocal resonance is increased over the area of diminished note. Crepitations are heard from the left apex to the level of the sixth spine, and when the left arm is placed on the right shoulder these crepitations are found to extend along the vertebral border of the left scapula. At the extreme right apex the percussion note is diminished, expiration prolonged, and a few crepitations are heard.

Pulse 80 regular. Heart sounds good. No murmur.

Tongue moist, a little flabby. Appetite excellent. Bowels opened daily. No vomiting or pain after food.

The temperature did not rise above 99° F. (37·2° C.).

November 17.—He complained of marked increase in intensity and extent of the pain on the left side of the chest. Wide-spread superficial tenderness. Headache and scalp tenderness.

The cough is more troublesome, expectoration no longer contains blood.

The right side of the chest remains as before, but there is a marked outburst of râles below the angle of the left scapula and in the left axilla.

Has lost his appetite. Feels sick after food. No diarrhœa.

November 26. Much pain on left side of the chest with superficial tenderness over the areas in the figure. Headache more marked on left side than right, and accompanied by marked scalp tenderness. (Fig. 22).

His appetite has completely gone. He feels sick and has vomited after food. The tongue remains clean. Bowels opened daily. Motions show no marked alteration.

The crepitations over the left base and in the left axilla have considerably increased since November 17.

Temperature reached 99·8° F. (37·7° C.) every night.

By December 20 all referred pain, superficial tenderness and headache had disappeared. He was taking his food well, and the nausea and vomiting were absent.

The left side of the chest behind was dull from the apex to the base. Over the upper part of the left lung behind expiration was prolonged and vocal resonance much increased, but below the

angle of the left scapula the breath sounds were very feeble, and vocal resonance was diminished. A few scattered râles were heard over the left base No active signs in right lung. Heart's apex beat was normal.

Thus, the dyspeptic symptoms in this case came on with the implication of the left base, and disappeared when the base of the left lung became permanently affected.

(C) *True Gastric Dyspepsia.*

In the third type of gastric disturbance met with during the course of the invasion of the lung by phthisis, the referred pain and superficial tenderness bear no relation to the condition of the physical signs in the lungs. This is a true dyspepsia of gastric origin. The pain and tenderness extend over the sixth, seventh, eighth and ninth dorsal areas to a greater or less extent, but there is a marked tendency to the simultaneous development of referred pain and tenderness within the fourth cervical area. Now, this association of tenderness over the gastric areas of the trunk, with the areas about the clavicle and shoulder joint, is a common feature of true gastric disorders.

Pain after food with occasional vomiting, especially in the morning (apart from coughing), flatus, and the regurgitation of fluid into the mouth, are symptoms of this form of dyspepsia. The tongue is usually more or less furred, frequently with a red tip, sometimes it is soft and tooth indented, the bowels are always disturbed, constipation alternating with attacks of diarrhœa, the evacuations are frequently made up of small hard lumps of dark fæces with some leathery curds and darkish brown yellow fluid, and are distinctly offensive.

This form of dyspepsia is more directly amenable to treatment directed towards the gastro-intestinal tract, and a considerable number of the cases are improved by the ordinary rhubarb and soda mixture accompanied by the occasional use of cascara sagrada.

Thus, the distribution of the referred pain and tenderness, the condition of the tongue and evacuations, the flatus and regurgitation of fluid into the mouth, and the results of

treatment, all point to some disturbance of the stomach itself as the cause of this type of dyspepsia in phthisis.

Case 70.—*Dyspepsia of gastric origin, accompanied by referred pain and superficial tenderness, arising in the course of a case of phthisis.* Bernard McD. (V.P.H., Dr. Harris); aged 21, cook.

In May, 1892, he was serving in the Marine Artillery, when he broke his ankle. For this he was invalided, and he spent the

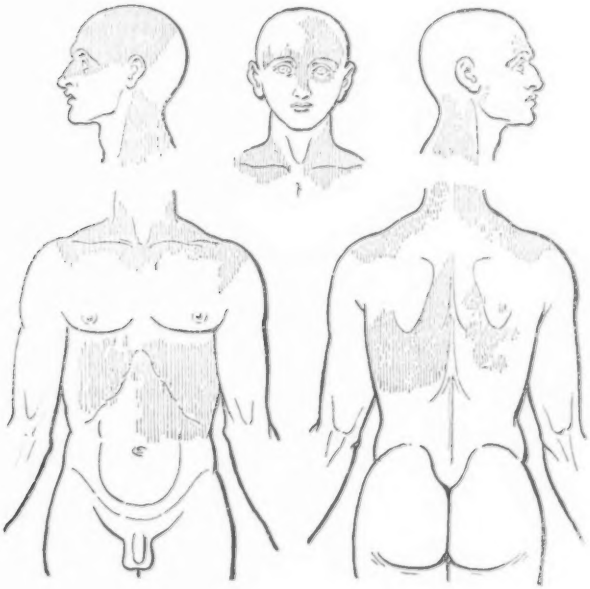


FIG. 23.

To show the areas of superficial tenderness accompanying an attack of dyspepsia, of true gastric origin, arising in the course of a case of phthisis [No. 70]

next five months nursing a friend, who died of "consumption of the lungs and throat." He then began to feel languid and faint, and "caught cold after cold."

August, 1893.—Cough came on badly; he began to sweat at night, and lost 7 lbs (3·4 kgm.) in weight.

February, 1894.—Hæmoptysis. Continued to bring up blood in small quantities for weeks.

Family History.—He has been entirely separated from his family for four years. One sister (who is in a convent) has had repeated hæmorrhage "from the lungs," and is dying of "consumption."

Admitted to V.P.H., July, 1894.

He suffered much from pain, vomiting, and alternating constipation and diarrhœa; coming on in attacks at varying intervals. The condition on September 7 was typical of several attacks which were watched during his stay in hospital.

September 7, 1894.—He is a well-built man of 5 feet 10 inches. Face thin; and there is much wasting about the neck. He has lost 3 stone (over 20 kgm.) in twenty months. No marked sweating at night. Face pale, and of a uniform earthy white. No flush. Lips pale, but distinctly blue. Temperature ranges to 101° F. (38·3° C.) at night, but sinks to 98° F. (36·7° C.) in the morning.

He has been somewhat freer of gastric disturbances until today. Tongue somewhat blue-red, moist; over the posterior part of the dorsum there is a triangular area of brown fur. Just before dinner he has a feeling of hunger and emptiness. Then he feels sick, and occasionally vomits. An hour after food, this same sinking feeling comes on again. He is passing green, very offensive, motions, three or four times in the twenty-four hours.

He complains of much pain, particularly in the left side, both back and front, from about the level of the sixth rib to the level of the umbilicus. It is most marked in the epigastrium, and behind the angle of the scapula. He also has pain in the posterior aspect of both shoulder joints, running up the back of the neck, and causing a feeling as if his neck was stiff. The pain is sharp, and leaves behind it a kind of "benumbed, painful feeling." Then, when he moves, the sharp pain comes on again within the "benumbed areas."

Headache over the forehead and temples. Hair is sore when brushed.

Marked superficial tenderness of body and scalp, as in fig. 23.

This pain is always more or less present now. It is worse before food; is somewhat relieved by food; and is worse again about an hour after a meal. Some flatulence; and the pain is easier when he brings up the wind.

Respiration 20. Sterno-mastoids acting. Very short of breath on exertion. Cough in occasional paroxysms; especially in the morning, when the cough causes vomiting. Expectoration in irregular, greenish muco-purulent masses, but not very profuse. (It has been tinged with blood since admission).

The left side of the chest is now dull from top to bottom, both back and front. Expiration is prolonged, and vocal resonance increased all down the left side, and in left axilla; and many crackling râles are heard. (The left base became affected on August 18, and has remained in the same condition ever since).

Over the right side of the chest the note is impaired as low as

the fourth rib in front, and the fifth spine behind. Over this dull area the breath sounds are bronchial. Vocal resonance is increased, and numerous fine crackling râles are heard. (The right side only was affected on admission in July.) The right base around and behind the angle of the scapula, and the lower part of the right axilla, are clear.

Palpitation and feeling of faintness on exertion. Pulse 88; small, regular. No abnormal sounds in the heart.

Urine, specific gravity 1022, acid, and with some albumen.

He died at the beginning of November, 1894.

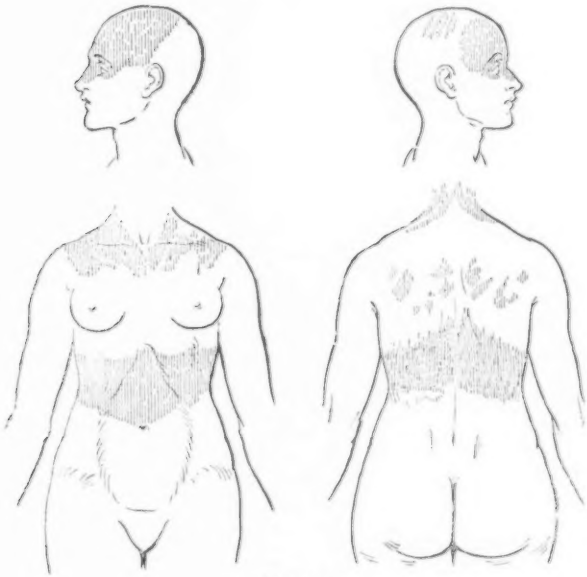


FIG. 24.

To show the areas of superficial tenderness accompanying an attack of dyspepsia of true gastric origin, arising in the course of a case of phthisis [No. 71].

Case 71.—*Dyspepsia of gastric origin, accompanied by referred pain and superficial tenderness, arising in the course of a case of phthisis.*—Minnie E. (V.P.H., Dr. Harris); aged 23, waistcoat maker.

In the winter of 1888-89 she began to have a dry, harsh cough, which passed off with the coming of the spring.

October, 1889.—She began to suffer from pain after food, and much vomiting. With this attack a hard cough came on again.

She was admitted to a general hospital, and there told she had a gastric ulcer. (Her abdomen still shows the scars of the frequent blisters that were then applied to the epigastrium.) She was well in ten weeks.

June, 1890.—The cough came on again, with nausea and pains in the limbs. Said to be influenza. In five weeks she was better, but very weak. August, 1890, a second similar attack, from which she did not recover for a long time.

Every winter the cough reappeared, but cleared off with the summer, until 1893. The cough then became constant.

September, 1893.—She began to waste rapidly, and to sweat profusely at night. The cough became much looser.

February, 1894. *On admission.*—Very marked purple flush upon the cheeks. Marked wasting. Sweats at night.

Respiration 35 to 40. Sterno-mastoids acting vigorously. Much subjective dyspnœa. Single troublesome cough. Brings up a large quantity of stringy aerated mucus.

Much pain at epigastrium and under shoulders, especially on the left side. Marked superficial tenderness over the sixth and seventh dorsal areas, with spotty tenderness over the fourth cervical area, on the left side. Widespread fronto-temporal headache, with superficial tenderness over the frontal mid-orbital, fronto-temporal, and temporal areas of the scalp.

Right apex moves better than the left. Percussion note diminished over left front. Over the upper two spaces on left side, in front, breath sounds are cavernous; bronchophony is audible, and many crackling râles are heard as low as the fifth intercostal space. Over the first two spaces on the right side the breath sounds are bronchial, and bronchophony is present, and numerous crackling râles are heard.

Behind: on left side, diminished note as low as level of fourth spine; breath sounds cavernous, with whispered bronchophony. Crackling râles from apex to around angle of scapula, and in the axilla. Over the right side: note diminished, as low as sixth spine. Over upper lobe: bronchial breathing and bronchophony; but opposite the level of third and fourth spines, an area of profound cavernous breathing and whispered pectoriloquy.

Pulse 100; small, regular. Apex beat, fifth space, internal to nipple line. Cardiac dulness slightly diminished. No abnormal sounds.

Tongue somewhat dark red, and not well moistened. Appetite poor. Bowels opened two to three times daily.

Liver and spleen not felt. Nothing abnormal felt in the abdomen.

Temperature sometimes touched 103° F. (39·4° C.) at night, and did not fall below 100° in the morning.

The signs on the left side became quieter, but the right side progressed rapidly. The pain and superficial tenderness became more marked on the right side.

At the end of March the gastric symptoms became a much

more prominent feature, and the superficial tenderness was very marked.

April 30.—She is vomiting green watery fluid in the morning, apart from cough, and before breakfast. The tongue is dry and red, with some irregular whitish fur, but not heavily coated. Appetite is very bad. She has been constipated, but the motions are now three or four in the twenty-four hours. The evacuations consist of some irregular brownish lumps, with much brown offensive liquid material.

She is complaining of much pain in the epigastrium and under the shoulder blades. She also has pain over the neck on both sides and over the shoulder joints. Very marked superficial tenderness, as in fig. 24. Headache over the temples and forehead, with superficial tenderness, as in fig. 24.

Liver and spleen not enlarged. Nothing abnormal felt in the abdomen. No marked signs of dilatation of the stomach.

The signs in the lungs have advanced rapidly. *In front*, there is dulness, with cavernous breathing and whispered pectoriloquy, as low as the fourth rib on the left side. On the right side, hollow breath sounds and whispered bronchophony, as low as third rib. *Behind:* on left side there is dulness to level of sixth spine, with cavernous breathing and whispered pectoriloquy to level of third spine. On the right side, dulness, with cavernous breathing and whispered pectoriloquy, as low as angle of left scapula.

The temperature does not vary more than about a degree, and the mean temperature is 101° F. (38·3° C.).

This case illustrates the true gastric disturbance of phthisis, and shows the association of the cervical areas of tenderness with those of the epigastrium.

It is frequently very difficult, in practice, to decide in which of these three groups to class some particular gastric disturbance that occurs during the course of a case of chronic phthisis. From a clinical point of view, such a division into three forms of dyspepsia would be scarcely necessary. But, from the physiological aspect, such a division is absolutely essential; for the three types are the expression of, at any rate, two fundamentally different processes.

Moreover, if we remember that two of these forms of dyspepsia are produced reflexly, whilst one is of gastro-intestinal origin, we can understand the following facts, upon which most observers are agreed. It would seem that in spite of the notorious proneness of phthisical patients in this stage of the disease to suffer from gastric disturbance,

in the large majority of cases no abnormal activity, either of the mucous membrane or of the muscular walls of the stomach, can be discovered. On the other hand, certain cases show distinct secretory and motor abnormalities.

Again, it helps us to understand why, in a large proportion of the cases, the dyspepsia tends to disappear, or to alter in type, as the destruction of the lung progresses. For the two purely nervous forms, due to the destruction of the bases of the lungs, must cease when the changes in the lung assume a form incapable of generating referred pain. Thus the "reflex dyspepsia" is a very prominent feature of the invasion stage of the disease, when organic changes in the walls and coats of the stomach are uncommon; whilst it may be entirely absent in the third stage, when alterations in the walls of the stomach are of much more frequent occurrence.

This assumption of a reflex origin for many of the dyspepsias, which appear during the invasion stage of phthisis, helps us to understand why gastric symptoms are not a marked feature of pneumonia, pleurisy, or of those cases of phthisis where the destruction of the lung is mainly by cavitation. For complete consolidation prevents the appearance of referred pain, by throwing the nerve endings of the diseased portion of the lung out of play. Cavitation destroys the nerve endings in the lung, and is, therefore, unaccompanied by referred pain. Pleurisy causes a local pain, in which the splanchnic system of afferent nerves is not implicated. Thus we should no more expect reflex dyspeptic symptoms to accompany a pure pleurisy than we should expect them to accompany an inflammation of the skin of the chest wall of similar extent.

To sum up the results to which we have arrived in this section:—

The gastric disturbances, which occur in the course of phthisis, may be divided, *clinically*, into three groups:—(1) The prodromal dyspepsia, appearing before there are any marked signs in the lungs. This dyspepsia is frequently accompanied by marked referred pain and superficial tenderness. It seems to be associated with the cachexia which not infrequently heralds the onset of the disease.

(2) The dyspepsia of invasion, also accompanied by referred pain and superficial tenderness.

(3) The dyspepsia of dissolution, accompanied by marked signs of organic change in the walls and mucous membrane of the stomach, but unaccompanied by referred pain and superficial tenderness.

Physiologically, the second of these clinical divisions can be again divided up into three types, according to the causes which give rise to it:—

(*a*) A pseudo-gastric disturbance, due to the nausea and vomiting produced by the referred temporal headache, which stands in definite relation to the referred pain produced by the implication of the base of the lung.

(*b*) Gastric hyper-æsthesia, which owes its origin to the fact that the bases of the lungs and the stomach are supplied from the same segments of the central nervous system. Thus an affection of the base of the lung causes a disturbance within the sixth to the ninth dorsal segments, and the stomach becomes hyper-æsthetic by sympathy, just as the testicle becomes tender in some cases of stone in the kidney.

(*c*) True dyspepsia of gastro-intestinal origin, having no relation to the nature of the phthisical process, or the distribution of the disease within the lung.

In conclusion, I have to offer my sincere thanks to the staff of the Victoria Park Hospital for Diseases of the Chest, for the extremely liberal manner in which they have permitted me to make use of their cases, not only during the time I acted as house physician, but also during the last three years.

My thanks are also due to Mr. Lunn for permitting me to make use of some of the cases under his care at the St. Marylebone Infirmary.

ON THE INFLUENCE OF REAGENTS ON THE ELECTRICAL EXCITABILITY OF ISOLATED NERVE.

BY AUGUSTUS D. WALLER, M.D., F.R.S.

(*Continued from p.* 67.)

BEFORE proceeding to review the effects produced upon isolated nerve by reagents in solution, we must consider the conditions of observation. We are acting on the hypothesis that a current of injury between longitudinal and transverse points of the nerve depends upon greater chemical activity at the transverse, or more injured point, and that a current of action, or negative variation of the injury current, depends upon a superadded chemical activity most pronounced at the longitudinal, or less injured part. We shall therefore apply our reagent to this less injured part, and watch for an alteration in the rhythmical series of deflections. Before we know the separate action of water, alcohol, salt, &c., it is obvious that we may not proceed to apply solutions to the nerve. Looking back to the result of *e.g.*, chloroform vapour, we may assume an increased deflection and a diminished deflection, as signs of excitation and of depression respectively. But the possibilities of chemical reaction between an added fluid and the clay electrode itself need to be remembered, although this is no very formidable danger, for we shall not often be obliged to run into it; alterations of the galvanometer zero thus produced do not interfere with our observations of the deflection itself, and after all we can easily test some extreme instances. Nor will the alteration of resistance due to adding fluid to an electrode cause us much trouble, for we can at any moment gauge such alteration by closure of a key to send from the compensator a small standard current into the experimental circuit.

At first this mode of trial was promising of results. A drop of saline solution had no effect on the deflection, a drop of water diminished it but very gradually, a drop of muscarin solution abolished it, so did a crystal of common salt, a drop of chloroform, a drop of 10 per cent. alkali, a drop of 10 per cent. acid; in all cases the reagent being restricted to the longitudinal electrode and superjacent portion of nerve.

But a little further reflection makes plain to us the unworkability of this working hypothesis. It will not do to apply a drug in solution locally to the nerve on the longitudinal electrode. That electrode serves as the lead-off not merely to the portion of nerve lying upon it but also to adjacent portions. Such portions may or may not have been reached by the drug, may or may not have been affected by it. The conditions of observation are, in short, nothing like so favourable as in the case of a gas or other volatile drug, which, when passed into the gas-chamber, equally influences the whole nerve.

This reflection impresses upon us the need of submitting the whole nerve to the action of the drug. We may lift off the nerve, let it soak in a watch-glass of fluid to be tested, and replace it on the electrodes. This seems at first sight a clumsy proceeding. Or we may lay the nerve on the electrodes clinging to a strand of wool moistened by salt solution, then apply our fluid with a pipette to the, so to speak, imbedded nerve, to all parts of which the wool acts as carrier of fluid by virtue of capillarity. This, at first sight, seems neater, involves no disturbance of contact, and we may feel sure that the whole of the nerve has been submitted to the drug.

We must, of course, under these, as indeed under any circumstances, exercise discretion in the comparisons we make and the conclusions we draw. To compare variations before and after a solution has been added, or before and after a wet thread has been laid along the nerve, and to attribute the difference to an action of the drug, would be absurd, for the resistance has been altered and additional derivation paths have been established. What we may do,

though, is to learn with certainty whether an effect has been suddenly abolished or suddenly augmented, and failing sudden action, to witness the gradual development of a change dating from the moment of interference.

And—provided all reserve be made in the adoption of conclusions—we shall, in case it be required to ring the changes in a succession of trials, prefer the simpler and easier method of taking off the nerve, soaking it for a definite time in the test solution, and replacing it on the electrodes as nearly as possible in its original position. It is, in fact, better to deliberately remove and replace the nerve, passing it with a glass rod through watch glasses of reagent and of saline, than to attempt to put a moist thread on and off, for the nerve is pretty sure to cling, and if more than one trial is to be made the electrodes must obviously become contaminated.

A precaution of principal importance is to test the resistance after each re-adjustment; it is evident that failing this precaution, we cannot be certain of the significance of an increased or diminished variation which might be due to a diminished or increased resistance, chiefly of the nerve itself, according to the fluid employed.

We shall attend principally to the rhythmic excitation effects, laying little stress upon any shifting of the zero that may have taken place. As a matter of fact, however, it is surprising, when we consider the delicate adjustment of the spot, how nearly its original position is recovered if a nerve is taken off, washed in salt solution, and replaced as nearly as possible *in statu quo*. Nor is there usually any great alteration of resistance, which implies that the variation before and after the bath are not on any very different scale, as is the case with the thread device.

Within what limits it may be safe to draw conclusions as to the action of a drug, will be best illustrated by an example.

The first drugs that I happened to use, not because I had much reason to imagine them to be active upon nerve, but partly because they were the drugs we tried together on the heart, partly in pursuance of the idea that muscarin is a

general protoplasm poison, and that nerve is a very simple sort of protoplasm, were muscarin and its antagonist, atropine. I took muscarin as being a drug with which I was most familiar as regards its action on the heart, where, as you may remember, we saw reason to believe that it is more correct to regard muscarin as depressant of protoplasm rather than as a specific excitant of inhibitory ganglia. And it was with this drug and with the same solution that you saw used in the series of heart experiments, that I went through certain doubts and difficulties which we may find it instructive to review. I will summarise them as quickly as possible by running through the records of their occurrence.

Plate 206[1] was my first trial of the possible action of muscarin on nerve. A drop of muscarin solution was placed upon the nerve where it rested upon the longitudinal electrode. Here is the essential part of the protocol, the plate exhibits an unmistakable effect of the drug.

Time.		Deflection.
0 mins.	before muscarin	16 (regular)
2 ,,	after muscarin	12
3 ,,	.. ..	8
4 ,,	.. ..	5
5 ,,	.. ..	3
7 ,,	.. ..	1
10 ,,	.. ..	?
15 ,,	.. ..	0
30 ,,	.. ..	0

This looked all right, but on the next trials (plate 210) no marked effect, if anything an increased deflection.

Before muscarin	15 (regular)
After muscarin	19 (regular)
After more muscarin	17 (regular)

[1] Many of the plates used in this lecture have not been reproduced here. Measurements only, or curves summarising several measurements are given of the chief significant experiments, with the exception of figs. 2, 3 and 4, which are records of the three most common bromides, and of figs. 9 and 10, in which an active and an inactive reagent (aconitine and aconine) are contrasted. These examples will suffice for the present to justify the application of the method to drugs in solution. Other records, inclusive of some that are referred to in this lecture, will be given in the next number of BRAIN.

Plate 211.—No marked effect.

Before muscarin	23
After muscarin	25 (falling to 22)
After more muscarin	17 (regular)

Plate 212.—Doubtful effect.

Before muscarin, 15.
After muscarin, 15 falling steadily to 8 during the next 25 minutes.

Plate 213.—Still more doubtful effect.

Before muscarin, 16 (regular).
After muscarin, 22, falling steadily to 17 during the next 20 minutes.

This was growing monotonous, these negative results were failures, which for the moment I could not account for. They were failures because the positive result of the first experiment seemed so clear and non-accidental (barring the one hardly probable accident of a contaminated glass rod used to put on the muscarin) that one could not admit the inactivity or the doubtful activity of muscarin. One might, perhaps, feel tempted to fall back upon some refuge formula for one's ignorance and say, as has been said in a similar case of doubtful results, "instability of nerve," but unstable is precisely what a nerve is not, and the use of such a formula concerning experimental results is a very thinly disguised and pretentious way of confessing ignorance.

But anyhow, the "failures" gave me pause, and led me to make reflections as to the correctness of strictly local application at a longitudinal electrode, and to try more extensive application of the drug by the soaked thread, or by actually bathing the nerve in a watch-glass. This cleared the matter up, and showed that the more extensive application of the drug was the proper mode of testing. I think, but of course cannot be sure, that the first positive and subsequent four negative results were owing to the fact that in the first trial, not knowing whether anything was to happen, I made very sure that the nerve should have its muscarin solution, and daubed it on freely, while in the next four trials, expecting an elegant effect, I applied a mere droplet with great care and neatness to the very centre of the nerve lying across the longitudinal electrode.

Forgive me for dwelling on such a trivial matter, it was a by no means unimportant trifle, inasmuch as the momentary difficulty helped to point out what I believe to be the right way of trying a nerve by a drug, and it is not an uninstructive illustration of a stumbling-block becoming a stepping-stone.

Plates 219-220.

Time.			Neg. Var.	Gradn. 0·002 volt.
0 mins.	Nerve only	.. ..	—	.. 11
1	,, ...		42	.. —
4	,, ...		42	.. —
5	,, Nerve + saline thread		—	.. 55
6	,, ...		13	.. —
7	,, ...		13	.. —
9	,, ...		13	.. —
12	,, ...		13	.. —
14	,, Nerve + muscarin thread		—	.. 60
15	,, ...		7	.. —
20	,, ...		5	.. —
25	,, ...		3	.. —
30	,, ...		0	.. —
31	,, Nerve + atropin thread		—	.. 60
35	,, ...		0	.. —
65	,, ...		0	.. —
95	,, ...		0	.. —

Plates 221-2-3.—Muscarin then atropin.

Time.			Deflection.	Gradn. 0·002.
0 mins.	Nerve only		15 (regular)	.. 10
5	,, Nerve + saline thread ..		8 (regular)	.. —
10	,, Nerve only		20 (regular)	.. —
15	,, Nerve + muscarin thread ..		— ..	.. 20
16	,, ...		9	
17	,, ...		7·5	
18	,, ...		6·5	
19	,, ...		5	
20	,, ...		3	
21	,, ...		2	
22	,, ...		1·5	
23	,, ...		1	
24	,, ...		trace	
30	,, Nerve + atropin thread ..		0 ..	.. 20
60	,, ...		0	
90	,, ...		0	

You naturally inquire next about atropin. So did I, and in the last two experiments with a negative result. This is

unsatisfactory. If the muscarin effect was a true physiological modification we should, from the analogy of cardiac muscle, expect that modification to be antagonised by atropin. But I must pass on, leaving this unsatisfactory result as it stands for the present[1]; there are other and far more satisfactory results that demand our attention, results which, however, were the immediate outcome of this check. And when you have realised what a rich harvest lies around us, I think you will forgive me for not remaining behind to gnaw this very tough and in itself probably not very nutritious root.

Once satisfied of the ease, simplicity, and clearness with which the action of a solution upon nerve can be tested we have but to go and take groups of substances as fast as we can think them out. I have already too much gathered material to be able to lay it before you in detail, but must, on the contrary, pick, and choose, and concentrate.

To that end I will put before you—

(1) Data relating to the haloid salts, with special reference to the bromides in particular.

(2) Data relating to some alkaloids and other hypnotics, taken almost at random.

(3) Data relating to antagonism.

Here are nine experiments showing the action of halides, all the experiments having been made under—as far as possible—constant conditions. In each case the nerve was bathed for one minute in a solution of decimolecular strength. Judge for yourselves whether the depressant action depends *more* upon the basic or upon the acid moiety,—in KBr, *e.g.*, whether the K or the Br is the predominant partner. And take note that on these data you can only judge whether one or other moiety has a predominant action; other data would be necessary before you could tell what (if any) is the share of the subordinate moiety in the total action. You may, indeed, make a shrewd guess at the facts of the case, from my use of the words "subordinate" and "predominant." And here is a

[1] I have not yet found time to repeat these experiments, and am still doubtful whether, or no, muscarin and atropin affect nerve fibres. In any case, their action (if any) is much less marked than that of *e.g.*, aconitin, and cocain and physostigmin.—(July, 1896.)

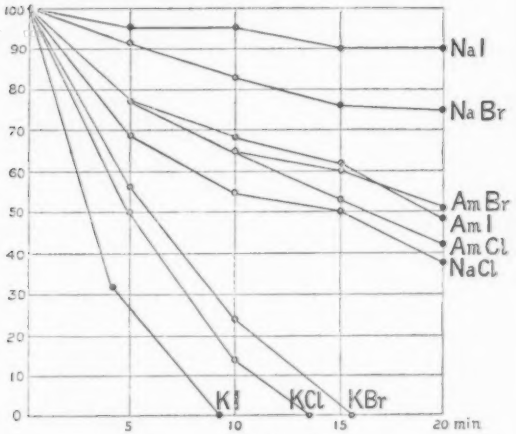


FIG. 1.

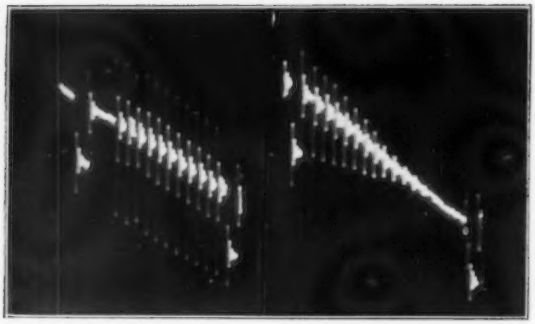
Curves exhibiting the effects of the chlorides, bromides and iodides of ammonium, sodium and potassium, upon the electrical excitability of isolated nerve.

In each instance the nerve, after its normal response had been recorded, was bathed for one minute in a decimolecular solution of the salt, and the response recorded for a further 20 minutes.

To construct the diagram, each of the nine series of measured records was reduced to a common measure, the normal response being taken as 100. The actual electromotive value differed in the different experiments of this figure, but may be regarded as approximately 0·001 volt.

	Chlorides.				Bromides.				Iodides.		
Plate No.	*(281)*	*(282)*	*(280)*		*(274)*	*(272)*	*(271)*		*(283)*	*(286)*	*(285)*
Normal	100	100	100		100	100	100		100	100	100
	AmCl	NaCl	KCl		AmBr	NaBr	KBr		AmI	NaI	KI
5 min.	76	68	50		75	91	55		75	95	32
10 ,,	65	55	12		65	82	22		68	95	0
15 ,,	53	50	0		60	75	0		61	90	0
20 ,,	41	37	0		50	75	0		48	90	0
25 ,,	30	27	0		—	—	—		—	—	—

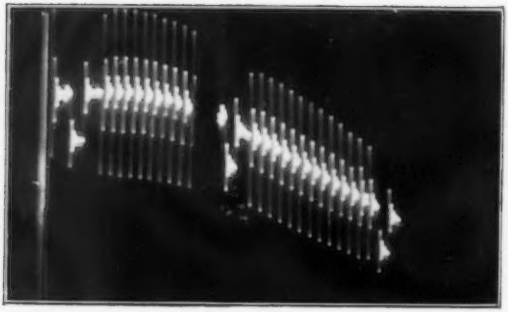
FIG. 2.



Before. ↑ After.

KBr. $^{m}/_{10}$.

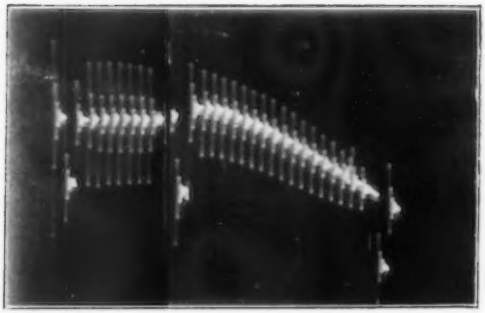
FIG. 3.



Before. ↑ After.

NaBr. $^{m}/_{10}$.

FIG. 4.



Before. After.

AmBr. $^{m}/_{10}$.

FIGS. 2, 3 AND 4.

Influence upon nerve of the bromides of potassium, sodium and ammonium.

In each case the nerve, after its normal response has been recorded for about 10 minutes, is bathed for 1 minute in a decimolecular solution of the bromide, and the response recorded for a further period of about 15 minutes.

The bromide of potassium produces most effect, that of sodium least effect.

single plate (No. 520), exhibiting the action of strontium bromide, which will suffice to prove that the basic moiety is not the sole efficient partner nor even always the predominant partner. NaCl, NH_4Cl, KCl are, as you remember, depressant salts; $SrCl_2$ is anti-depressant; K and Sr in the respective chlorides are the principal and opponent elements, Sr being, indeed, the most prominently anti-depressant element that has yet come under my notice in these experiments. Yet the compound $SrBr_2$ is, as you see, a depressant salt. As we shall see in a moment, the chlorides of strontium and of potassium are mutually antagonistic; the bromides of strontium and of potassium are not so.

The predominant influence of the basic moiety—in the present case the greater depressant action of potassium than of sodium—is illustrated in other instances; thus to name only such cases as I have experimentally verified, caustic potash is more active upon nerve than caustic soda, potassium carbonate than sodium carbonate, potassium cyanide than sodium cyanide, taken, of course, on an equi-molecular scale of solution.

On the other hand, the subordinate influence of the acidic moiety—not very marked in the series of salts we have just reviewed—is clearly seen in the case of the series EtCl, EtBr, EtI. There is a general resemblance in the action of these three compounds, they are anæsthetics of the ether type, and have each, at one time or another, been recommended as such in actual practice. But their effects are by no means identical: ethyl bromide is apt to be irritant; ethyl chloride gives an anæsthetic effect that is apt to be more fugitive than desired; ethyl iodide is apt to produce a lingering after-depression. These are, I believe, the kind of character that the three drugs have earned clinically, and volatility is no doubt a factor in the result, thus ethyl chloride has found its most extended practical application as a local anæsthetic in dentistry, on account of its volatility. But the point to which I am now inviting your attention is that the general action of ethyl varies with its acidic companionship. Fig. 2 is a summary of actual experiments, of which the measurements are

given below; and you will, perhaps, be inclined to recognise in the results there embodied indications towards the selection of anæsthetics that, in this case, at least, are in unison with the slowly acquired conclusions of clinical experience—I had nearly said experiment. Imagine, for a moment, that clinically untried drugs are before you—ethyl dioxide, ethyl chloride, ethyl bromide, and ethyl iodide, you would in all certainty, if you used these nerve experiments as your sign-post, follow the way that has been followed by practical anæsthetists, rejecting the bromide and the iodide, and adopting almost exclusively the ethyl dioxide as your ordinary anæsthetic.

The conclusion to which I have been led by the study of the action of salts upon nerve may provisionally be summed up in this succinct formula.

A salt AB, taking effect upon living matter, acts in its compound capacity AB, not in an individual capacity A, nor B, nor A + B; in such compound action, B is more effective than A.

Or, figuratively expressed, the action of the firm composed of A and B is that of the firm AB (where A's action is modified by B's, and B's modified by A's, giving a total action expressible as AB's) not that of the partner A, nor that of B, nor that of A plus that of B. In the total action AB, the influence of B predominates.

The action of KBr is not that of K only, nor that of Br only, nor that of K plus that of Br; it is the action of KBr as a whole, but an action in which the characteristics of K are most distinctly recognisable. K is the predominant, though not solely, effective partner. KCl, KBr, KI, are not of identical effects, nor in the anæsthetic series are C_2H_5Cl, C_2H_5Br, and C_2H_5I; but in the two series it is the basic element potassium and the basic radicle ethyl that characterise the two groups; all three members of the first are depressant; all three members of the second are anæsthetic.

Just one remark touching the bromides, which, as you well know, are prominent among the calmatives of the *materia medica* in correction of nerve exuberance, such as epileptic and other convulsions. The bromide of potassium

is *the* calmative bromide, but in cases where this bromide is found to be too depressing, those of sodium, or ammonium, or lithium, or calcium, or strontium, are prescribed in its place. I have tested all these bromides upon nerve; of all the effects are similar in character, though differing in degree; there is no augmentation by $CaBr_2$, or by $SrBr_2$. Evidently these bromides may be tolerated when KBr is found to be too depressing. But I must take some other occasion for discussing this bromide question in detail.

Here are records showing the antagonism between members of the sodium group and members of the calcium group—an antagonism that was first made known to us by Ringer's experiments with the frog's heart.

A few words of orientation are desirable here.

Physiological antagonism pure and simple should consist in this: an impulsion towards greater or smaller activity due to one set of molecules, neutralised by another set of molecules acting in the opposite sense. And an antagonism in its truest sense should be *simultaneous* antagonism, so to speak, a tug of war between two opposed sets of molecules acting simultaneously upon the protoplasm of contention.

As ordinarily studied, the antagonisms physiologically examined, such as muscarin-atropin, eserin-atropin, potassium-calcium, &c., have been *successive* antagonisms; the effect produced by a given reagent has been abolished by the subsequent administration of a second reagent.

Towards the formation of clear conceptions in pharmacodynamics, this distinction between simultaneous and successive antagonism should I think be made, and we need not stop to discuss the cases in which, owing to unequal rates of diffusion, two simultaneously applied reagents might successively come into effective action.

The distinction drawn between simultaneous and successive antagonism will help us to distinguish between what may be termed "true" and "false" antagonism. In a true successive antagonism, the physiological effect of one reagent is abolished by a second reagent of opposite effect, independently of any mere physical removal or chemical

neutralisation of the first reagent; in a false antagonism, the first reagent is, so to speak, actually turned out by the second, or chemically neutralised, just as salt is washed out of a sponge by water, or an acid is neutralised by an alkali. Such effects should, I think, be excluded from our notion of physiological antagonism.

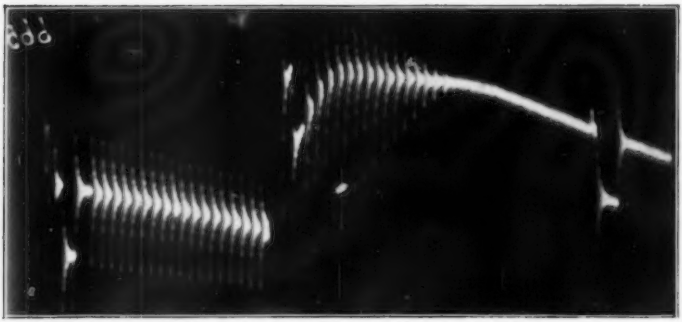
Here, then, are three cases on nerve which may serve to illustrate my remarks.

The first is a case of *false antagonism*. (Plate 256). In consequence of a one minute bath in a semi-molecular solution of NaCl (2·9 per cent.), the excitability of the nerve has declined. In consequence of a subsequent one minute bath in distilled water, the excitability of the nerve has been temporarily restored. We shall not, on that account, designate NaCl and H_2O as physiological antagonists. NaCl has been washed out by H_2O.

The next is a case of *successive antagonism*. In consequence of a one minute bath in a decimolecular solution of $SrCl_2$ the excitability of the nerve has been augmented. Half-an-hour later, with the strontium augmentation still present, the nerve was immersed for one minute in a decimolecular solution of KCl, in consequence of which excitability rapidly declines. This rapid decline is not a mere effect of removal of strontium; under similar conditions, but with decimolecular NaCl in place of KCl, it did not take place. I may add that the Sr *versus* K antagonism is mutual; this particular nerve was a little revived by a second strontium bath, and I have other records of a Sr increase after K decrease.

Here, finally, is a case of *simultaneous antagonism*, in which, after having found that dubious results ensued from immersion of the nerve in a mixture of equimolecular antagonists (equal volumes of decimolecular KCl and $CaCl_2$), I bathed a nerve for one minute (*a*) in a solution composed of six parts $CaCl_2$, and four parts KCl; and (*b*) in a solution composed of four parts $CaCl_2$, and six parts KCl. In the first case Ca gets the best of it, and the series of effects is on the increase. In the second case K gets the best of it, and the series of effects is on the decrease.

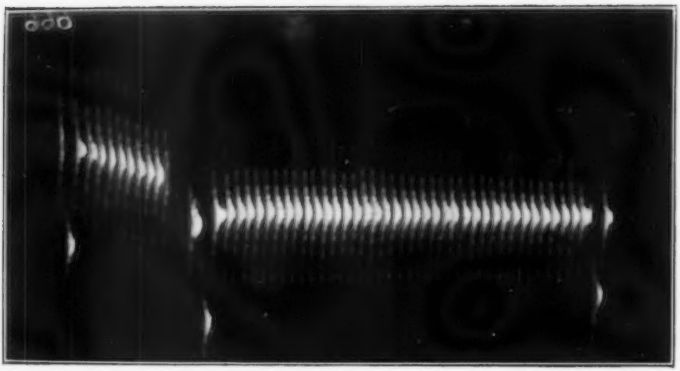
Fig. 9.



Before. After.

Aconitine 0·33 °/₀.

Fig. 10.



Before. After.

Aconine 1 °/₀.

Finally, let me again direct your attention to some alkaloids. We have already had something to say to muscarin and atropin; but as regards definiteness of result, the choice, or rather, the accident that led us to test these drugs first, was by no means encouraging; the result did not come out black and white, but a dirty grey. Further orientation experiments with other alkaloids have been far more satisfactory, yielding us results of the kind required in an early stage of investigation, definitely positive and definitely negative. Let me, however, at once guard the statements that I shall have to make as regards the negative action of a drug; they are comparative, and valid only under the conditions of time of immersion and strength of solution adopted in these experiments. We shall speak of a drug as having little or no action when its 1 per cent. solution has little or no effect upon a nerve immersed in it for a period of one minute, but such a statement must not be taken as committing us to a denial of any action whatever by the drug acting in stronger solution or for a longer period.

One of the best black and white contrasts that has presented itself has been between aconitine and aconine, the latter you may, for the moment, regard as being chemically a fragment of the former. Subject to what has just been stated, *i.e.*, under our conditions of experiment, aconitine promptly abolishes the excitability of nerve; aconine has no such effect. In order to bring out with any distinctness the gradually developing effect of aconitine, its solution must be taken below 1 per cent., at which strength the nerve is usually killed on the spot. Fig 9, and its curve in fig. 6, exhibit the effect of a one-third per cent. solution of aconitine, viz., the response of the nerve is at first exaggerated, subsequently abolished, somewhat as happens when the drug is applied to the excised heart. Fig. 10, and its curve in fig. 6, exhibit the comparative inertness of aconine.

These are chiefly orientation experiments, and I shall not enter into much detail at present, but shall simply bring before you a selection of such preliminary experiments, made with a view of ascertaining whether a given drug is to be characterised as active, or as inactive, in relation to the

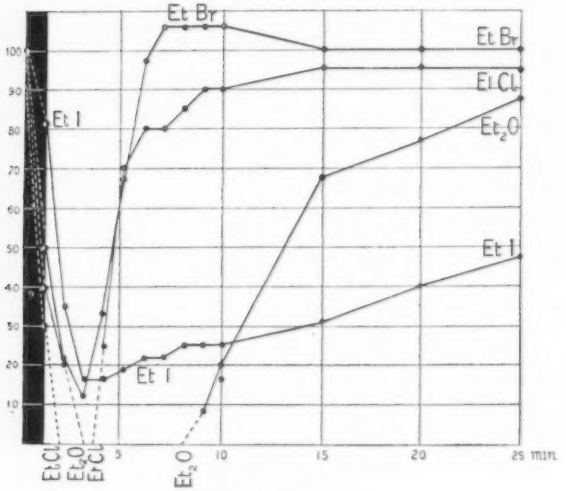


FIG. 5.

Curves exhibiting the anæsthetic effects upon nerve of ordinary ether (Et_2O), and of the chloride, bromide and iodide of ethyl (EtCl, EtBr, EtI).

In each case the nerve, after its normal response has been recorded for about 10 minutes, has undergone the influence of the anæsthetic vapour, blown through the nerve-chamber for 1 minute; the record has been continued for a further period of about 20 minutes.

TIME.				
0 min.	90	100	100	100
5 „	95	100	100	100
10 „	100	100	100	100
	Et_2O	EtCl	EtBr	EtI
1 min.	50	30	40	81
2 „	20	0	21	34
3 „	0	0	12	16
4 „	0	25	33	16
5 „	0	70	67	19
10 min.	20	90	106	25
15 „	68	95	100	31
20 „	77	95	100	40
25 „	88	95	100	47
30 „	95	95	100	52

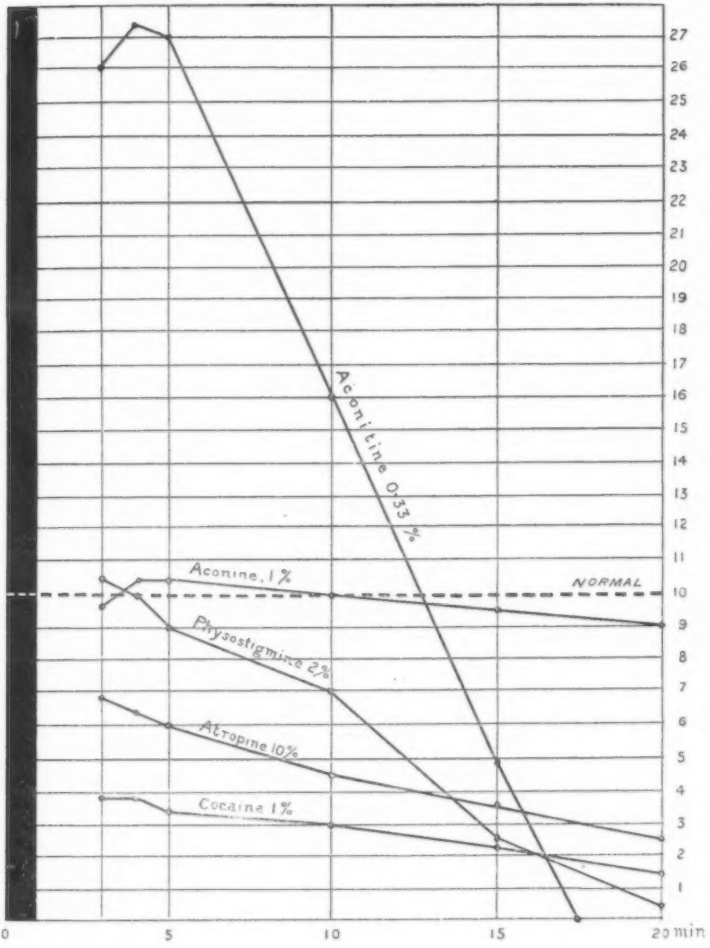


FIG. 6.

Curves exhibiting the effects upon nerve of:

(665)	Aconitine hydrochloride	0·33	per cent. solution
(666)	Aconine hydrochloride	1·00	,, ,,
(355)	Physostigmine sulphate	2·00	,, ,,
(3,129)	Atropine sulphate	10·00	,, ,,
(840)	Cocaine hydrochlorate	1·00	,, ,,

electro-mobility of the nerve. The rule of procedure has been to take for about ten minutes, at intervals of one minute, the response of the undrugged nerve, then to immerse the nerve for one minute in a given solution, then to resume the observation on the now-drugged nerve for a further period of twenty to thirty minutes. The actual records are laid out for your inspection on that side-table; our review of the

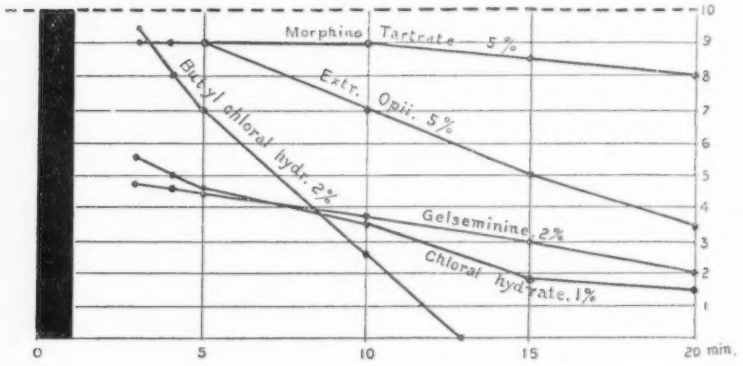


FIG. 7.[1]

Curves exhibiting the effects upon nerve of:

(567)	Morphine tartrate	5	per cent. solution.
(724)	Extract of opium	5	" "
(308)	Gelseminine	2	" "
(321)	Chloral hydrate	1	" "
(287)	Butyl chloral hydrate	2	" "

results will be facilitated and accelerated by these diagrams (figs. 6, 7 and 8), in which the measurements taken from the records have been reduced to a common measure—the normal response of the undrugged nerves being taken as 100, and its alterered response after drugging, indicated, therefore, by rising or falling percentage lines, time after drugging being

[1] In the experiments from which figs. 6, 7 and 8 have been constructed, the nerve, after its normal response had been taken for about 10 minutes, was removed from the electrodes, bathed in the solution named for 1 minute, and replaced on the electrodes. The deflections seen on the actual records at beginning and end of series of responses (figs. 2, 3, 4, 9 and 10) are standard deflections by an electromotive force of 0·001 volt. In the curves of figs. 5, 6, 7, and 8, and in their tabulated measurements, the reduction to a common measure (normal = 100) has been carried out as in the curves of fig. 1. But in order to indicate whether the modification of the nerve by a drug has been made on a series of increasing, decreasing, or constant magnitude, the normal point of departure = 100, has been taken at the end of the normal series of responses, just before the nerve has been drugged, and the measurements given of 5 and 10 minutes before this point: *e.g.*, in fig. 9, there is a diminishing series before the nerve has been drugged, and the normal point = 100, has been taken at its end.

marked along the abscissa. With regard to these individual experiments, there is little more to be said than may be gathered on the face of the diagrams themselves, or of the records, if you care to examine them. There are one or two points, however, to which I may call your attention.

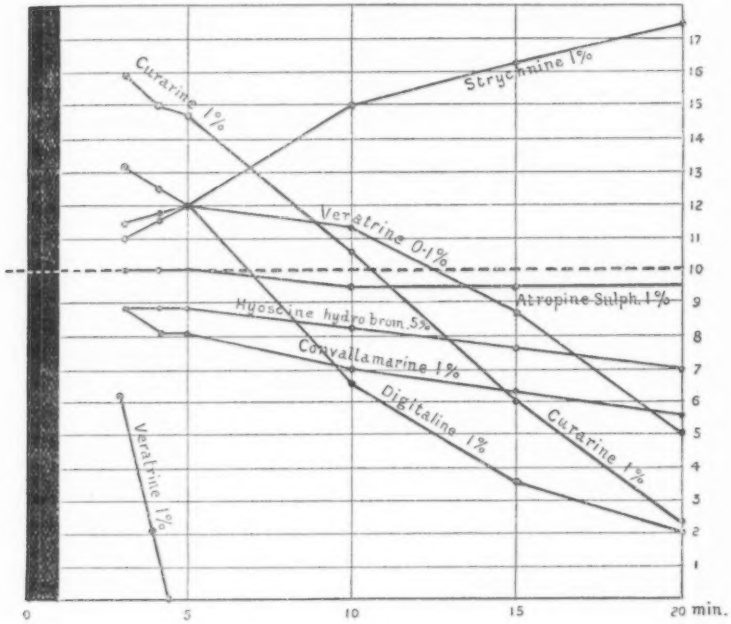


FIG. 8.

Curves exhibiting the effects upon nerve of:

(3,125) Curarine	1·0	per cent.	solution.
(3,128) Strychnine sulphate	1·0	,,	,,
(3,195) Digitaline	1·0	,,	,,
(3,196) Convallamarine	1·0	,,	,,
(3,201) Hyoscine hydrobrom.	5·0	,,	,,
(3,132) Atropine sulphate	1·0	,,	,,
(3,122) Veratrine hydrochlor.	1·0	,,	,,
(3,200) Veratrine hydrochlor.	0·1	,,	,,

Morphine (in this case in the form of tartrate, also I may add in that of the hydrochlorate; the bimeconate I have not yet examined) produces remarkably little effect. The observation used in the construction of the curve (I have

repeated it three or four times with a similar result) was made with a five per cent. solution of morphine tartrate.

It naturally occurs to one to ask whether the crude drug, from which morphine among other principles is obtained, is as inactive on nerve as morphine itself seems to be. Plate 724 (represented in the curve of fig. 7) gives the answer, and indicates as possible that some constituent of opium, other than morphine, is effective in the narcosis of nerve. The experiment has been repeated two or three times with similar results, but obviously requires development by other experiments.

Gelseminine, chloral hydrate, butyl chloral hydrate (which is clinically regarded as having a special predilection for the fifth nerve), and cocaine, tell their own story; but it is only with the last-named drug that I have made experiments in any number; and specimens of different source seem to act more or less forcibly, always however in the direction of depression. Atropine in 1 per cent. solution (fig. 8) has little or no action, in 10 per cent. solution (fig. 6) seems to have acted; but is it fair to put a drug in such an exaggerated concentration upon an isloated nerve, and then say that it acts upon nerve? I think not. Hyoscine (fig. 8), which is of the same family with atropine, is hardly less inert as regards isolated nerve, although clinically it is an undoubted and most potent sedative. One naturally reflects on possible differences of effect, according as a drug encounters grey cerebral matter by channel of the circulation, or medullated matter by direct application; some drugs are general anæsthetics through the brain, others are local anæsthetics at their seat of application; that is the empirical fact with which we must be content, and concerning which I know of no plausible explanation. Veratrine, like aconitine, is a local anæsthetic, and like it, is very active upon the nerve; in 1 per cent. solution the response is promptly abolished (see fig. 8); in 0·1 per cent. solution even, the response, after undergoing augmentation, suffers fairly rapid diminution.

Of all the curves plotted out in figs. 6, 7 and 8, the only one that exhibits prolonged and steady increase is that of strychnine; further experiments will, however, be required

before I shall venture to conclude that this is a regular effect. The observation is an isolated one, and the effect may therefore have been accidental.

Two other drugs that I have tested have given regular results, as indicated by the curves in fig. 8. Curarine—to a physiologist, one of the most interesting of drugs; to a chemist, not yet possessing any legal status—is distinctly active. Digitaline—clinically, of the greatest importance as a cardiac tonic; but chemically, figuring under many disguises—has likewise a distinct local action upon the nerve.

Convallamarine, allied to digitaline as regards its action upon the heart, has a similar but less pronounced action upon the nerve (in fig. 8).

These are, I am well aware, in relation to the magnitude of the subject, very fragmentary results. So far, however, from offering an apology for introducing the matter to you in such unfinished fashion, I am disposed to claim credit for so doing, inasmuch as contact with a growing surface of knowledge, physiological or otherwise, is the most valuable lesson that a teacher is able to offer.

You now know at least as much about the matter as I do; you have taken part in the preliminary difficulties of technique and in the avoidance of fallacies. As you know, I had no idea where we were going, and least of all any utilitarian anticipations, when I set up apparatus to show you a presumably delicate experiment. I used to think it a ticklish matter to exhibit without fail the negative variation of nerve; it has turned out to be easy. You have seen it going by the hour; you have also seen how easy it is to determine on nerve the quantitative ratio between the stimulus and the effect; and, finally, how satisfactory a test-object nerve affords as regards the unambiguous results of experimental interference. The apparatus (galvanometer, coil, rheostats, condenser, &c.) are, comparatively speaking, delicate apparatus, but by no means so delicate that they may not be "minded" by ordinarily skilled hands. Then I wanted to show you the *autographed* results of such observations, and you have seen how this was done with

the roughest possible apparatus—an old soap-box with a horizontal saw-cut; an ordinary photographic quarter-plate carrier fastened to a bent pin at the end of a thread, and let down by a wheel fastened to the minute axis of a common American clock. Nor was the gas-chamber a very complicated or expensive piece of apparatus. It would cut a very sorry figure in a modern laboratory or in an instrument maker's catalogue.

Yet a familiarity with such common things has not, I am certain, bred contempt in you for the questions that we have attacked together, but rather a curiosity for further knowledge, and a sense of its accessibility by simple means, and by "constantly thinking unto it."

Just consider what a vast field of knowledge lies open to us ready to be explored by this clean and keen little weapon —a tag of frog's nerve. I meant more than an evil-tasting joke in saying that a frog's nerve conducted itself like an ordinary man when subjected to carbon dioxide, to alcohol, and to tobacco smoke. I also had chloroform and ether in my mind, their glaring difference on nerve, the glaring difference in their death-rates when used as anæsthetics. Will you have ether or chloroform? Will you, as in America, use ether whenever you can, or as in England draw up death-rate statistics of chloroform, and appoint commissions to debate whether deaths by chloroform are by the heart or by the lungs? Then turn your thoughts to the pharmacodynamics of living matter; where may not these little tags of nerve lead us along its intricate paths? I do not know, but I feel myself entitled to repeat the exclamation used by my father in 1850, when he had found that the nerve fibre was kept good by its nerve cell—"A cloud as small as a man's hand."

Yet as observations are multiplied, and with them the number of exceptional or anomalous results, one may not resist a feeling of considerable perplexity. Question after question has unmasked itself from behind each new experiment; and one may well feel oppressed by the extent of possibilities before us, oftener than contented by the few scanty facts hurriedly surveyed.

I am not sure whether muscarine and atropine are to be put down as active or not on nerve-fibres. I only know that they have no activity comparable with that of cocaine or aconitine. I am not sure whether the antagonism between potassium and calcium is true or false. I have sometimes failed to obtain augmented action by strontium and by calcium, and I do not know what may be the conditions of the appearance or non-appearance of their effects.

Unfortunately, for the moment—yet no doubt fortunately in relation to the ultimate issue—the simple case of a nerve with a current of injury giving a negative variation when excited, is by no means simple, when its elementary explanation is pressed for. We have to study other cases of excitatory effects and after-effects, positive effects and positive after-effects, as well as negative effects of excitation, and the effects of reagents upon these. And then we shall find ourselves in a maze of possibilities, involving the study of electrotonic currents, of polarisation increments, and again, effects of reagents upon all these. We shall have to wade through many perplexities and complications before we reach to any degree of elementary simplicity, and, if I do not forbear exhibiting this damping outlook to you, it is because I wish you to put their proper value upon simple and easy ideas, and to realize that if they are easy when found, they are difficult to find and to make easy. I do not know where we may be going from this point, but the clues that I intend to pursue are certain suppositions relating to simultaneous negative and positive phenomena in nerve, connected with its polarisation and depolarisation, and belonging perhaps to the general doctrine of dissimilation and assimilation that we have already considered in connection with retinal phenomena. And the reagent, from the minute study of which we may feel most hopeful of further information, is carbon dioxide; it is the prominent dissociation product of living matter. We have seen that it profoundly modifies the excitability of living matter; the first part of our task will therefore be to study its effect upon the polarisation and depolarisation of medullated nerve.

Time.	(No. 321).	(No. 287).	(No. 3,125).	(No. 3,128).
0 min.	100	100	94	100
5 ,,	100	100	97	100
10 ,,	100	100	100	100
	Chloral hydrate. 1 % for 1 min.	Butyl chloral hydrate. 2 % for 1 min.	Curarine. 1 % for 1 min.	Strychnine sulphate. 1 % for 1 min.
13 ,,	55	96	155	111
14 ,,	50	83	150	111
15 ,,	45	69	145	120
20 ,,	35	28	105	150
25 ,,	20	0	60	162
30 ,,	15	0	25	175

Time.	(No. 3,195).	(No. 3,196).	(No. 3,201).	(No. 3,132).
0 min.	100	112	100	100
5 ,,	100	106	100	100
10 ,,	100	100	100	100
	Digitaline. 1 % for 1 min.	Convallamarine. 1 % for 1 min.	Hyoscine hydrobrom. 5 % for 1 min.	Atropine sulphate. 1 % for 1 min.
13 ,,	130	85	90	100
14 ,,	125	80	90	100
15 ,,	120	80	90	100
20 ,,	65	70	82	95
25 ,,	35	63	76	95
30 ,,	20	56	70	95

Time.	(No. 665).	(No. 666).	(No. 355).	(No. 3,129).
0 min.	123	100	110	100
5 ,,	112	100	105	100
10 ,,	113	100	100	100
	Aconitine hydrochloride 0·33 % for 1 min.	Aconine hydrochloride 1 % for 1 min.	Physostigmine sulphate 2 % for 1 min.	Atropine sulphate 10 % for 1 min.
13 ,,	260	95	105	—
14 ,,	275	105	100	65
15 ,,	270	105	89	60
20 ,,	160	100	50	45
25 ,,	47	95	22	35
30 ,,	0	90	5	30

Time.	(No. 840).	(No. 567).	(No. 724).	(No. 308).
0 min.	100	100	94	85
5 ,,	100	100	97	92
10 ,,	100	100	100	100
	Cocaine hydrochlor. 1 % for 1 min.	Morphine Tartrate 5 % for 1 min.	Ext. Opii. 5 % for 1 min.	Gelseminine 2 % for 1 min.
13 ,,	30	—	—	—
14 ,,	30	—	89	—
15 ,,	25	90	89	45
20 ,,	21	90	70	37
25 ,,	18	85	50	30
30 ,,	15	80	36	20

Time.	(No. 3,122).	(No. 3,200).
0 min.	96	96
5 „	98	100
10 „	100	100
	Veratrine hydrochlor. 1 % for 1 min.	Veratrine hydrochlor. 0·1 % for 1 min.
13 „	62	114
14 „	20	116
15 „	0	120
20 „	0	111
25 „	0	86
30 „	0	50
35 „	0	30

THREE CASES OF NEW GROWTH WITH CAVITY FORMATION IN THE SPINAL CORD.

BY WILLIAM ALDREN TURNER, M.D., F.R.C.P.

AND

ASHLEY W. MACKINTOSH, M.D.

We have been enabled, by the kindness of Dr. Ferrier, to record three cases of tumour of the spinal cord, which at one time or another during the course of the disease had been under his direct observation, either in King's College Hospital or at the Queen Square Hospital, and Cases 1 and 2 were exhibited and commented upon by him before the Neurological Society. The pathological examination of these cases has been carried out in the neuro-pathological laboratory at King's College, and we are indebted to Dr. Ferrier for the opportunities afforded us for this investigation.

Case I.—*Almost complete paralysis, with wasting of arms. Paresis of legs. Profuse general perspiration. Prominence of lower cervical and upper dorsal spines. Dissociation of sense of temperature from senses of touch and pain, practically limited to pectoral girdle. Death within two years of onset. New growth and cavity formation in cord, implicating both posterior columns and both posterior horns.*

F. K., aged 18, a groom, was admitted to King's College Hospital under Dr. Ferrier, on April 5, 1890. The following is an abstract of notes taken at that time and during the subsequent course of his illness:—

There was nothing worthy of note in his family history, or in the patient's previous history.

Patient was quite well till about the end of February, 1890,

when his shoulders and upper arms began gradually to get weak, the right arm more quickly than the left. At the end of three weeks he could scarcely raise his arms at all. There was no history of any blow or fall, or exposure to cold and wet.

Condition on admission.—He is a healthy-looking, well-nourished lad. There is great weakness in abduction, flexion and extension at both shoulders; while adduction is well performed. The shoulders droop downwards and forwards, and are very feebly shrugged. The action of the pectorals and latissimi dorsi is feeble. Flexion and extension at the elbows are weak, but at the wrists fairly strong. Flexion of the fingers is weak and the grip feeble. The movements of the legs show normal power. In walking, his head droops forwards, and his gait is shuffling. Movements of the face are normal. The electrical reactions of all muscles are normal. The knee-jerks are increased. There is no ankle clonus. Patient can feel and localise correctly. The hands are congested and cold, and the face sweats a great deal. The cervical spines are abnormally prominent, but there is no pain on pressure over them.

July 14.—The loss of power in the arms has much increased. There is now complete loss of movement at both shoulders and elbows, while flexion and extension of the wrists and fingers are weak, especially on the right side. He cannot abduct the second and third fingers. There is great wasting of all the scapular muscles, including the serratus magnus on both sides. The hands are cold and flushed. The face is flushed and still sweats. Occasionally there is a feeling of "pins and needles" in the feet, and pains in both arms. The legs have been feeling rather stiff for a few days. Once, while walking, patient felt a pain in the right leg along the external saphena vein, and the leg gave way, and he fell. From this time onwards, the legs have become weaker, especially the right.

October 2.—The hands are cold and livid. There is no power of movement in either arm, except weak supination of the right forearm, and feeble extension of the fingers of both hands. All the paralysed muscles react to faradism, the pectorals and triceps of both sides rather feebly. There is no reaction of degeneration. In all muscles K.C.C. is greater than A.C.C.. The left pupil is dilated.

January 30, 1891.—Patient was admitted to Queen Square Hospital under Dr. Ferrier. He states that his gait has gradually become worse, till now he cannot walk without assistance. For some time he has had gradually increasing difficulty in swallowing,

and occasionally some difficulty in passing water, but never incontinence.

Present condition.—There is some weakness of the abdominal muscles. He cannot raise himself up in bed, or sit up when raised. In deep inspiration the upper part of the thorax is elevated, but there is very little expansion. There is a small amount of movement at both shoulders and elbows, but practically none at either wrist-joint. There is some power of flexion and extension of the fingers, especially of the left hand, and chiefly of the thumb. The left arm is rather rigid. The movements of the legs are fairly good when in bed, but there is weak dorsal flexion of the ankles when standing up. Both legs are somewhat rigid, especially the left. The gait is feeble. He cannot walk without assistance, and drags both legs very much. He sways when standing with feet close together, and especially when his eyes are shut.

All muscles of arms and legs react normally to faradism, except that the re-action of the supra-spinati and infra-spinati is feeble. All muscles react normally and readily to galvanism, but the forearm-muscles show less than the normal difference between K.C.C. and A.C.C.

Sensation of all forms is normal, except that thermic sensibility is deficient on both arms from the elbows downwards. Sense of position of limbs is normal. The knee-jerks are equal and much exaggerated; there is no ankle clonus. No wrist or elbow-jerk can be obtained on the right side, but there is a distinct elbow-jerk on the left side. No plantar reflex can be elicited.

Trophic Changes.—There is marked wasting of both deltoids, and of the infra-spinati and supra-spinati; but no distinct wasting of the pectorals, trapezii or latissimi dorsi. The legs are not wasted. The face is flushed. Patient always feels hot, and his whole body is constantly bathed in sweat. His hands very quickly get blue and cold when exposed. There is a marked projection backwards of the spinous processes from the seventh cervical to the fourth dorsal spine, but no pain on pressure or on movement. There is slight nystagmus on lateral deviation, especially to the right. All ocular and pupillary movements are normal. The pupils are dilated, and the left is rather larger than the right. Swallowing, especially of solids, is somewhat difficult.

July 10.—The left pupil is much larger than the right. There is considerably more movement in both arms, especially the right.

Sensation.—Tactile sensibility is normal everywhere; but there is decided impairment of thermic sensibility on the neck and upper part of the trunk, extending down almost to the level of the umbilicus, and on both arms, especially the right.

October 23.—*Sensation.*—Tactile and algesic sensibility is still intact, but there is no appreciation of either heat or cold on the neck and trunk down to rather above the level of the umbilicus. The same is true of the whole of the right arm, and of the left arm down to the elbow, while there is imperfect appreciation of temperature on the left arm below the elbow. Below the area of thermo-anæsthesia, on the trunk, there is a zone where cold is appreciated, but not heat. All the muscles of the arms react to a moderate faradic current, but the deltoids require a somewhat stronger current than the other muscles. All the muscles react normally and readily to the galvanic current. The arms are rather weaker again. The left pupil is still larger than the right.

December 3.—There is still profuse general perspiration; the face is flushed and markedly injected, the hands are livid and cold to the touch. The wasting of the shoulder-muscles is now very great, especially on the left side. There is marked wasting of the interossei and of the thenar and hypothenar eminences. There is still some power of flexion and extension of the fingers, but only very slight movement at the wrists and elbows. No movement is possible at the left shoulder, except what is due to the pectoralis major, while there is a fair power of adduction, but no abduction at the right shoulder. There is still a considerable amount of power in the legs.

Both knee-jerks and plantar reflexes are enormously exaggerated, especially the right, and there is well-marked ankle clonus on both sides. There is incontinence of urine and fæces, but patient is conscious of the necessity. There is obstinate constipation. Nystagmus is now distinct in both eyes on lateral deviation. The pupils are now equal and dilated, but react normally. The function of all other cranial nerves is normal.

Tactile sensibility is normal everywhere, except that it seems rather defective towards the lower edge of the ramus of the jaw; and there is on the anterior aspect of the trunk a well-defined semi-lunar area of anæsthesia, the upper margin of which passes from nipple to nipple, about one inch below the xiphoid, and the lower margin through the umbilicus. The legs are hyperæsthetic.

Algesic sensibility is everywhere normal.

Thermic Sensibility.—From the chin anteriorly, and a little below the line of hair posteriorly, down to the level of the umbilicus both anteriorly and posteriorly there is complete thermo-anæsthesia; except that on the area above the second rib in front, extending up between the sterno-mastoids, all thermic sensations are called cold, and there is a slight appreciation of heat and cold

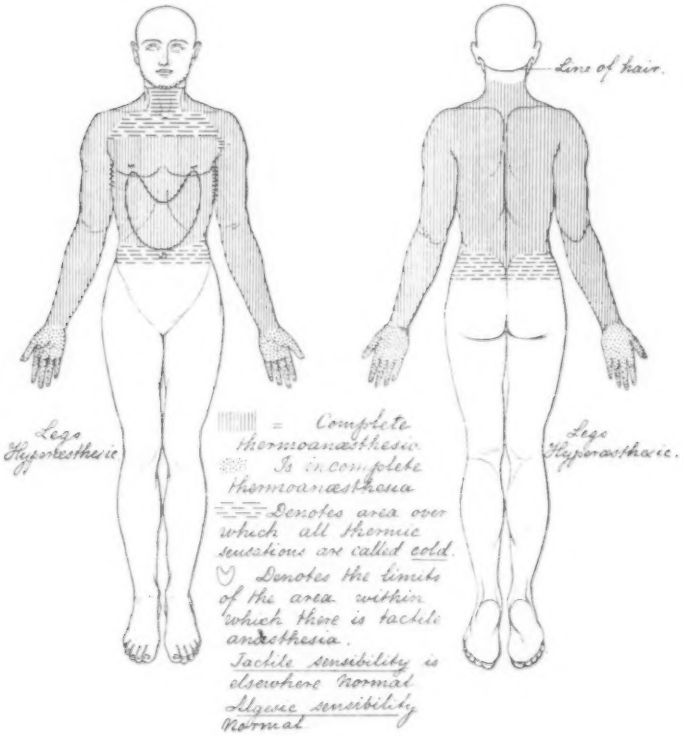

Line of hair.
Legs Hyperæsthesic
= Complete thermoanæsthesia
= Incomplete thermoanæsthesia
Denotes area over which all thermic sensations are called cold.
Denotes the limits of the area within which there is tactile anæsthesia.
Tactile sensibility is elsewhere normal
Legs Hyperæsthesic.
Muscle sensibility Normal

CHART 1.

in both axillæ. Below the area of thermo-anæsthesia on the trunk, there is a zone about two inches in depth on which all thermic sensations are called cold. Below this, the trunk and legs have normal thermic sensibility. On the arms there is complete thermo-anæsthesia down to the wrists, while there is diminished appreciation of temperature on the hands

Muscular sense is normal in all the limbs.

January 14, 1892.—Patient began to suffer from difficulty in breathing a week ago. His temperature rose, and he became much weaker, and died to-day.

There was influenza in the wards.

At the *autopsy* it was found that there was no angular curvature of the spine. The cord in the cervical region was enormously enlarged, and filled practically the entire vertebral canal—the medulla appeared to be implicated in the enlargement. In the upper part of the cervical region the cord was found, on section, to consist almost entirely of a dark pulpy mass, surrounded by a mere rim of nerve tissue. The amount of central new growth gradually diminished lower down, and, at the various levels of the cord, presented the same relations and the same extent of cavity formation as will be found in the detailed description of the microscopical appearance of the sections of the cord.

Microscopical Examination of the Spinal Cord.

Mid-cervical Region (fig. 1).—At this level the cord measures 21 m.m. transversely, by 16 m.m. antero-posteriorly. Each posterior column in its anterior two-thirds is occupied by a new growth, composed of laminæ concentrically arranged; the structures of the cord in the neighbourhood are much compressed, so as to cause an alteration both in shape and position. The grey commissure is retained. The lumen of the central canal is still present; its ventral side is lined by several layers of endothelium, which, from the form of the cells and their method of staining, look like normal lining cells; but the dorsal side of the canal is entirely bereft of the normal lining, and the lumen is broken into by a large number of round and spindle-shaped cells. In some of the sections from this region there is, dorsally and laterally to the central canal, a small area containing an enormous increase in the number of cells, amounting actually to a third tumour. The intermediate grey matter is much broken up, especially on the left side, due, apparently, to infiltration of gliomatous tissue. The anterior horns are normal and unaffected by the tumour-growth, the large ganglion cells presenting normal features. The left posterior horn is compressed, but not directly implicated by the new growth; the right posterior horn is distinctly involved in the growth, and in it there lies a cavity surrounded by gliomatous tissue.

Each tumour is composed of a slightly fibrillated neuroglial tissue, with few cells in its central part, but distinctly more

cellular towards the periphery; the cells are round or spindle-shaped. In the centre of the left tumour mass a recent hæmorrhage has taken place, while in the right tumour there are several small cavities, each containing a homogeneous fibrinous material, evidently the remains of a hæmorrhagic clot. These cavities, as well as that in the right posterior horn, are smooth-walled, but there is no trace of an endothelial lining. In the peripheral parts of each tumour, and in the adjacent nerve tissue, there is a great increase in the number and size of the capillary blood-vessels and of the smaller arterioles.

There is very marked degeneration in the whole of the antero-lateral region of the cord on the left side; while on the right side there is slight degeneration in the crossed pyramidal tract, extending forward in the antero-lateral periphery of the cord to the region of the direct pyramidal tract, where the degeneration is much more marked.

Lower Cervical and Upper Dorsal Regions (figs. 2 and 3).—The appearances in these two regions blend with one another, and therefore a continuous description is given.

(1) In the first of the sections at this level (fig. 2), the tumour-mass in the left posterior column is again seen, but reduced in size, and retaining its circular appearance. The definite tumour-like appearance on the right side has disappeared, and is replaced by an elongated oval tract of nerve tissue, apparently occupying the anterior two-thirds of the zone between the posterior horn and the posterior column, extending into the grey commissure, and containing a slit-like cavity partially filled with a fibrinous material. In the posterior half of the left posterior horn there is another slit-like cavity, surrounded by a thin layer of new tissue; while anterior to this, and in the most ventral and external part of the posterior column, there is another slit-like cavity also filled with fibrinous material, its anterior extremity passing into the grey commissure. The walls of all these cavities are very sharply cut, but in none is there a trace of an endothelial lining. The intermediate grey matter is broken up, especially on the left side, and in the spaces the same fibrinous material is seen. The central canal is represented by a mass of small round cells.

(2) In a section a little below the last, exactly the same appearance is seen, except that the tumour-mass in the left posterior column presents a clear-cut cavity in its centre.

(3) In this section, a little lower still, four cavities are now seen (fig. 3)—one in the posterior part of each posterior horn, and

one in the anterior part of each posterior column. Each of the cavities is very clearly cut, and is surrounded by neuroglial tissue, very small in amount, except around the cavity in the left posterior column, which is enclosed in a thick, dense mass of neuroglial tissue. In this region, the intermediate grey matter seems to be undergoing myxomatous degeneration.

There is degeneration in both crossed pyramidal tracts, in the antero-lateral periphery of the cord, especially of the right side, and in both direct pyramidal tracts—degeneration is specially distinct in the last.

Mid-Dorsal Region.—At this level, the cavities are limited to the posterior horns, one on each side. They present the character of clean-cut cavities, surrounded by a dense layer of neuroglial tissue without a trace of an endothelial lining; by their anterior extremities they extend well into the intermediate grey matter, and posteriorly they reach almost to the tip of the gelatinous substance of Rolando. The cavity on the left side contains homogeneous fibrinous material.

Lower Dorsal and Lumbar Regions (figs. 4 and 5).—In all sections made in these regions, the same features are observed—viz., a single cavity, surrounded by a layer of neuroglial tissue, in the anterior two-thirds of the left posterior column, close to the posterior horn, but distinctly separated from it by nerve fibres. The only difference at the several levels is the shape of the cavity. At the level of the eighth dorsal nerve roots, the cavity is an antero-posterior slit; in the lower dorsal region (fig. 4) it has a spur-like process extending outwards; while in the lumbar region a similar process passes inwards as well, so that the cavity presents a crucial appearance. In the lower lumbar region (fig. 5) there is the same appearance, with the addition of a small localised gliosis, occupying quite the anterior part of the left posterior column; this is separated from the cavity by a well-marked strand of nerve fibres. In one of the sections this gliosis shows clear signs of cavity formation. In all these sections the central canal is represented by a mass of small cells, and the posterior commissure is normal. In the mid-dorsal and lower-dorsal regions there is well-marked degeneration in the pyramidal tracts of both sides, direct and crossed; in the lumbar region the degeneration is limited to the crossed pyramidal tracts.

We are indebted to Dr. James Taylor for the description of the macroscopical appearance of the cord, and for the sections from which the microscopical examination was made.

Remarks.

In this case, points of clinical interest are the comparatively rapid course—death taking place, within two years of the onset of symptoms—and the dissociation of the sense of temperature from the senses of pain and touch.

The new growth and cavity formation, as figs. 1-5 show, involve the grey commissure, the intermediate grey matter, the posterior horns and the anterior two-thirds or so of the posterior columns, especially in their external parts. Fig. 1 shows two distinct tumour-like masses in the posterior columns, that on the right side implicating the posterior horn as well. From the concentric arrangement of the laminæ in the new growths, and from the fact that the grey commissure is present, it appears likely that the growths originated in the posterior columns, especially in their external parts, or in the adjacent posterior horn.

There is a direct transition from the true tumour-like formation in the mid-cervical region (fig. 1) to the appearance presented in figs. 2 and 3. The tumour-mass on the right side in fig. 1 is converted into the mass of gliosis, seen on the right side in fig. 2 occupying the same region, viz., the external part of the posterior column and the adjacent part of the posterior horn, while, in a section made a little lower (fig. 3), this same region shows two smaller masses of gliosis, each with a clear-cut cavity in its centre, the posterior lying in the middle of the posterior horn and the anterior occupying the region between the anterior part of the posterior horn and the posterior column. Lower still, about the mid-dorsal region, the gliotic process on the right side is strictly limited to the posterior horn, and gradually ends there, without showing at any part a direct communication with the region of the central canal.

Similarly, the growth seen on the left side in fig. 1 loses its distinct tumour-like appearance, but more gradually than on the right side: thus in fig. 2 it is still quite like a tumour, and there is now a distinct cavity in its centre, continuous with the cavity formed by the hæmorrhage in fig. 1. But there are also two pieces of gliosis on the left side in fig. 2,

each with a clear-cut cavity, both in the posterior horn or adjacent part of the posterior column, the anterior reaching well into the grey commissure, but not communicating with the region of the central canal. In fig. 3 the growth in the left posterior column has now lost the tumour-like appearance, and consists of a thick mass of gliosis, with a very clearly-cut cavity in its centre; there is now only one cavity in the left posterior horn, corresponding in situation with the posterior of the two cavities in fig. 2. Still lower, in the mid-dorsal region, the gliosis and cavity formation on the left side become limited to the posterior horn; while in the lower dorsal and lumbar regions the morbid process moves gradually towards the adjacent part of the posterior column, leaving the posterior horn intact (figs. 4 and 5). There is absolutely no trace of an endothelial lining to any of the cavities, despite their remarkably clear-cut appearance. At no part is there a dilated central canal; it is represented in almost the whole dorsal and lumbar regions by a mass of nuclei.

The gradual and direct transition between the true, tumour-like appearance, or *gliomatosis*, in the sense of Hoffmann,[1] seen in fig. 1, and the appearance presented in fig. 3, is very striking; the latter presents all the signs of a gliosis or proliferation of the neuroglia. There can be no doubt that the corresponding cavities are continuous throughout.

The fact that a fibrinous material is found in many of the cavities, points to a hæmorrhage as their cause, and there is an undoubted hæmorrhage on the left side in fig. 1. At other parts there is an appearance of myxomatous degeneration, leading to cavity formation.

Attention may be drawn to the fact that the morbid process, when traced to its lower limit, ends, not in the region of the central canal, but either in the posterior horn (as on the right side, *cf.* also Case 2), or in the adjacent part of the posterior column (as on the left side); so that if any deduction can be drawn from the mode of termination as to the starting point of the process it

[1] Hoffman. *Deutsche Zeitschr. f. Nervenheilk*, 1893, iii., s. 1-136.

would be against the view of a central origin (*i.e.*, an origin around the central canal), and in favour of the view already drawn from the appearance presented by fig. 1, viz., that the growths started in the posterior column, or adjacent parts of the posterior horns.

We have been unable to trace any connection between the small mass of gliosis in the anterior part of the left posterior column in the lower lumbar region (fig. 5), and the larger gliosis behind it, or the gray commissure in front of it, so that it seems quite an isolated piece of gliosis.

The symmetrical affection of the two sides of the cord, and the bilateral descending degeneration, are in accordance with the fact that the symptoms were bilateral and symmetrical; this presents a contrast to Case 3, in which it will be seen that the lesion and the symptoms were mainly unilateral.

CASE II.—*Almost complete paralysis with wasting of arms. Paresis and rigidity of legs. Hyperidrosis of face and arms. Marked pain and tenderness in upper part of spine. Loss of sensation over pectoral girdle, at first thermo-anæsthesia only, to which was added later analgesia, and finally anæsthesia: legs slightly affected before death. Death within one year of onset. New growth (neuroglioma ganglionare), and cavity formation in upper part of cord, involving mainly posterior columns and posterior horns.*

A. P., aged 23, a 'bus-driver, was admitted into King's College Hospital, under Dr. Ferrier, on June 5, 1894. The following is an abstract of notes taken at that time and subsequently:—

Family History.—Patient's father and two brothers died of heart disease, and an aunt had a stroke of paralysis. No tubercular history.

Previous History of Patient.—Patient has been much exposed to cold and wet. There is an indefinite history of an injury to the spine ten years ago. Three years ago patient had "influenza," and suffered from occipital pains for six months afterwards. No specific history.

History of Present Illness.—About the end of February, 1894, on getting off his 'bus one night, patient was seized with severe

pain between the shoulders. His neck felt stiff, and he had difficulty in straightening his back and in using his arms. This condition passed away in about an hour, but ever since that time, patient has had attacks of pain shooting from his neck down both arms and up to his occiput, and he has complained of stiffness of the neck, and of a feeling of "pins and needles," and numbness in the region of the pains.

Condition on Admission.—Patient is a well-nourished, healthy-looking man. He holds his head and neck very stiffly, and any forcible attempt to move them causes pain. Under an anæsthetic there is found to be some resistance to movement of the head to the right. All movements of the arms are present, but weaker than normal. He can stand quite steadily with eyes shut. The knee-jerks are present and equal. There is no ankle-clonus.

SENSATION.—*Subjectively*, there is a burning pain round the right side of the upper part of the chest, and a sharp pain shooting down the right arm. *Objectively*, tactile and algesic sensibility is normal. There is tenderness on pressure over the upper part of the spine extending down to the second dorsal spinous process. The function of all the cranial nerves is normal.

After the above date, there was a temporary improvement in all the symptoms, especially the pains, but later the pains in his neck and arms and between the shoulders returned with increasing severity, and both arms began to get weak, the left one first. On August 15, 1894, patient entered University College Hospital, under Dr. Bastian. The following is an abstract of notes of his condition, made by Mr. C. Atkinson, and kindly supplied to us by the Registrar:—Movement of the head is limited in all directions owing to the pain caused thereby in the region of the cervical spine. Flexion of the trunk is limited. There is marked weakness of both arms, especially the left. At the shoulders the power of abduction is lost on both sides, flexion is lost on the left side, and limited to 30° on the right side, while rotation also is lost on the left side. At the elbows, there is some power of flexion on the right side, none on the left. Movement at the wrists and fingers is normal. The legs retain power of strong movement at all joints. The right knee-jerk is very brisk, but there is no ankle-clonus. *Sensation* of all forms is normal except that thermic sensibility is somewhat erratic on both hands, especially on the forefingers, and there is defective appreciation of heat over the upper part of the trunk; subjectively there is pain in the region of the upper dorsal spine, radiating down the right arm and up towards the occiput. There is marked hyperidrosis of face, hands and feet. The right pupil is larger than the left.

Subsequent Course.—The arms and legs became progressively weaker, the legs got rigid, the knee-jerks much exaggerated, and ankle-clonus well marked. Breathing became almost entirely thoracic, and occasionally the abdomen was retracted during inspiration. The pains continued very severe, but shifted their position lower down the dorsal spine. There was an almost constant feeling of extreme cold over the left shoulder and left arm down to the elbow, and over the left side of the trunk, and there was a marked girdle sensation round the chest below the level of the nipples. The spine remained tender on pressure, and the area of tenderness gradually extended down as low as the mid-dorsal region. The hyperidrosis of face and hands remained very constant. The muscles of the arms, forearms and hands gradually wasted to an extreme degree, but all retained a fairly normal reaction to the faradic current, although it was noted that A.C.C. was greater than K.C.C. as early as October 1. On October 29, a small circular ulcer, of the size of a split pea was noted over the head of the metatarsal bone of the right thumb, and a few days later a similar ulcer was found on the left thumb. Constipation remained obstinate throughout.

The Affection of Sensation gradually increased. Thus on September 9 it was noted that tactile and algesic sensibility was normal everywhere, while on the right hand up to the wrist, on the left hand up to the elbow, and on the chest from the third rib down to the costal margin *all* thermic sensations were described as cold.

The condition as regards sensation, was noted as follows, on October 11.

Tactile sensibility appears normal, except that patient cannot readily discriminate different forms of tactile impression.

Algesic sensibility is considerably blunted, but not lost, over the front of the chest, from the second intercostal space to the costal margin and over both arms, except over the outer part of both upper arms.

Thermic sensibility is defective over the whole of both arms, and over the trunk, from the level of the second intercostal space to the umbilicus.

The condition of sensation on October 31, was the same as in the last note, except that the area of defective algesic and thermic sensibility now extended on the trunk, down as far as Poupart's ligament, and the right arm had now quite lost the sense of pain, while thermic sensibility was quite gone on both arms.

On November 24, when patient left University College Hospital,

it was noted that tactile sensibility seemed distinctly deficient over both arms, and over the anterior part of the chest, while there was an extension of the complete loss of thermic and algesic sensibility to the upper part of the trunk, and there seemed to be a slight defect in appreciation of difference of temperature in the legs.

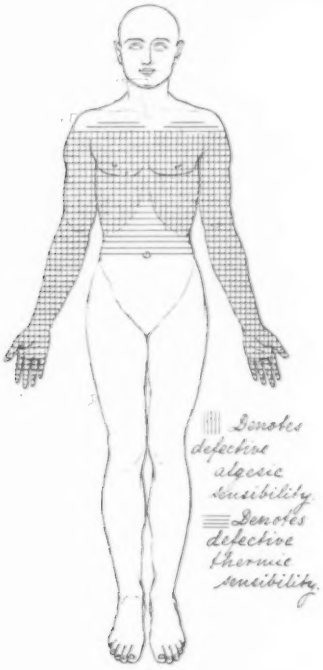


CHART 2.

The general condition of patient on re-admission into King's College Hospital on November 26, was as follows:—There is still marked hyperidrosis of face and arms, and subjective sensation of cold over the arms and abdomen. There is practically no abdominal respiration, and breathing is often very difficult. There is no power of movement in either arm, except some elevation of the shoulders, and some flexion and extension of the right wrist, and of the fingers of both hands. The legs are drawn up and rigid;

flexion at the knees is very weak, and movement of the ankles and toes is quite lost, except some slight movement of the halluces. All the muscles of the arms are much wasted; but all react to faradism, although the reaction of the deltoid, pectoralis major, triceps, and supinator longus of both sides is feeble. The knee-jerks are much exaggerated, especially the right; ankle clonus is well marked. There is constant pain down the whole length of the spine, but especially over the cervical and upper four or five dorsal spines; the pain is increased on movement, especially on sitting up in bed. There appears to be a slight irregularity of the upper two cervical spines. Movement of the head to the right is very limited on account of the pain. *Sensation* is as noted on November 24; the sense of position of the arms and fingers is not clear. The right pupil is rather larger than the left, and the tongue deviates slightly to the left. The function of other cranial nerves is normal.

December 13.—Sensation of touch is distinctly defective on the trunk, above the umbilicus.

Patient's condition remained unchanged till January 27, 1895, when his breathing suddenly became laboured; loud râles were heard in the chest, breathing got worse and pulse weaker, and he died on January 28.

At the *autopsy* it was found that the spinal cord was enormously enlarged and abnormally soft in the cervical region, filling practically the entire vertebral canal. The medulla did not appear to be implicated. The vessels of the dura mater of the cord were greatly distended. On section, the upper and middle cervical regions appeared to be quite filled with a soft, pulpy, reddish-brown mass, which bulged out at the cut ends. This abnormal appearance gradually diminished lower down, becoming more and more confined to the central region and posterior columns, till about the mid-dorsal region, where the cord appeared normal.

Microscopical Examination of the Spinal Cord.—The MEDULLA appears perfectly normal, except that it is abnormally vascular, especially in its posterior half, and at its lower part there is a distinct small-cell infiltration underneath the ependyma of the fourth ventricle.

At the level of the second and third cervical nerves (fig. 6) the central part of the cord is seen to be occupied by a new growth, which is circumscribed with remarkable clearness. It occupies the anterior three-fourths, or more, of the posterior columns and the grey commissure. There is no trace of a central canal, nor can the anterior commissure be seen, *i.e.*, the tumour growth

comes right up to the antero-median fissure. The intermediate grey matter, and both posterior horns, are very much compressed, and are directly invaded by the new growth, which extends along the posterior horns to the periphery of the cord. The grey matter of the anterior horns is also very much compressed, and its cells are smaller and fewer than normal. Degeneration is fairly well marked in the periphery of both sides of the cord, in the region of the direct cerebellar tract, and of the antero-lateral tract of Gowers, but there is very little sign of degeneration in what remains of the posterior columns. The new growth consists of small neuroglia-like cells, round or rather spindle-shaped, with deeply stained nuclei, and numerous fine processes ; the cells are much more densely packed at the periphery than in the central part of the growth. A striking feature is the great amount of vascular development in the new growth, especially at its periphery, and in the nerve-tissue immediately outside it. The vessels are very numerous; and many have thick, homogenous walls which stain very poorly. The central parts of the tumour have undergone a metamorphosis, partly fatty, as shown by osmic acid, partly myxomatous, as shown by spaces containing clear, homogenous tissue.

At the level of the fifth cervical nerves (fig. 7), the new growth reaches its maximum in point of size, filling the whole cord with the exception of a marginal rim which is nowhere thicker than 1¼ mm. The structure of the new growth is the same as at the last level, and its relations are also unchanged, allowance being made for its increase in size. The anterior horns are now very much compressed, but the ganglion cells are still quite visible, and have fairly well-marked processes. The anterior fissure is greatly displaced, and very little can be seen of the posterior horns. There is no distinct tract of degeneration in the rim of nerve tissue.

At the level of the sixth and seventh cervical nerves (fig. 8), the abnormal tissue has now considerably diminished in amount, and is confined to the posterior commissure, the intermediate grey matter, and the anterior two-thirds of the posterior columns. Posteriorly there is no sharp line of demarcation between the new growth and the normal nerve tissue. The anterior commissure now appears normal, and the central canal is distinct, but its wall is rather broken up. The anterior horns appear normal.

There are distinct cavities in the new growth, two in the anterior half of each posterior column. Those on the right side have a sharp, sinuous margin, being lined by a layer of wavy

tissue containing many deeply-stained nuclei; they are filled with a fibrin-like material. The cavities on the left side present a contrast to those on the right side, in that they have not such sharply-cut margins, their walls present a broken-down appearance, and they do not contain fibrin-like material. There is no trace of an endothelial lining in any of the cavities. The new growth consists mainly of small cells similar to those seen at higher levels, but very conspicuous now are larger bodies seen throughout the whole of the new formation at this level, but most numerous in the region of the intermediate grey substance. They vary considerably in size and shape (fig. 9). The largest are about the size of the ganglion cells of the anterior horn. Many are round or oval, others are rather irregular in shape. Their nuclei are large and stain deeply, in some there are two or more nuclei. These bodies are surrounded by a narrow clear space, and no trace of fine fibrillary processes can be detected arising from them. They stain in quite the same manner as ganglion cells.

There is no distinct tract of degeneration at this level, but there is, perhaps, a diffuse hyperplasia of the supporting tissue of the whole cord.

At the level of the eighth cervical nerves, the amount of new growth has still further diminished, the cavities in the right posterior column have disappeared, and there is now only a very slight amount of new tissue in the anterior part of the right posterior column, quite cut off from the region of the central canal. There is still a cavity in the anterior third of the left posterior column, surrounded by abnormal tissue which is connected with the posterior commissure. Both posterior horns are normal.

At the level of the first dorsal nerves the condition is the same, except that the gliotic tissue surrounding the cavity in the anterior part of the left posterior column is now quite cut off from the grey matter of the cord by normal nerve fibres, so that the posterior commissure and central canal region are quite normal.

Lower down still, the cavity in the left posterior column elongates antero-posteriorly, and tends to go more and more outwards till it invades the left posterior horn as well as the adjoining part of the posterior column, and the right posterior horn, which was quite normal at higher levels, presents a distinct nuclear infiltration which increases as we descend and develops a cavity in its interior.

At the level of the third dorsal nerves, there is a cavity on

either side strictly confined to the posterior horns, and not connected with the grey commissure; they are surrounded by a very small amount of nuclear tissue. These cavities gradually diminish lower down, till about the level of the fourth or fifth dorsal nerves, both cavities and abnormal tissue have disappeared, and the posterior horns and posterior columns become normal. In the upper and middle dorsal regions, but not in the cervical or lower dorsal region, the central part of the grey matter, and, to a less extent, the adjacent white matter show by Marchi's method of staining a vast number of black masses and dots (fig. 10), each larger mass being a conglomeration of smaller particles. Carmine-stained sections at the same levels show homogeneous colloid-like masses varying in size, and corresponding very closely in size and situation to the black masses seen by Marchi's method. There is no appearance of a nucleus in them. At these levels also, sections stained by the Weigert-Pal method, show that the grey matter is markedly deficient in the normal fibrillary nerve plexus.

From the level of the mid-dorsal region downwards, there is no abnormal tissue or cavity formation anywhere, and there is no sign of descending degeneration. The cord, in fact, seems perfectly normal. We shall make some remarks later as to the condition of the central canal.

Remarks.

The rapidity of the course—death within one year of the onset of symptoms—the marked tenderness of the cervical spine, the severity and persistence of the pains in the back of the neck, and shooting down the arms, and the progressive affection of the different forms of sensation—temperature, pain, touch, in the order named—are important clinical points in this case.

The enormous size which the new growth has attained at its maximum in the mid-cervical region, as shown in figs. 6 and 7, and the great amount of compression and distortion of the cord are very striking, especially when taken in conjunction with the fact that there is no distinct trace of secondary descending degeneration in the lower parts of the cord.

The part of the cord occupied by the new growth is the same as in Case 1 (*i.e.* the central region, the posterior

horns and the front of the posterior columns), but in this case the growth extends forward to the antero-median fissure (figs. 6 and 7) so that there is no trace of an anterior commissure.

On being traced downwards, the morbid process gradually assumes the appearance of a gliosis rather than that of a tumour, and becomes limited to the anterior parts of the posterior columns, while the region of the central canal becomes normal. The gliosis on the left side tends to move more and more outwards, so as to implicate the posterior horn, and finally it is restricted to the posterior horn where it ends about the level of the fourth or fifth dorsal nerves. On the right side the gliosis in the posterior column gradually ends at the level of the first dorsal nerves, but here the right posterior horn, which appeared quite normal at higher levels, begins to show a small cell infiltration, which gradually increases as we descend, develops a cavity in its centre, and ends in the posterior horn at the level of the fourth dorsal nerves: this piece of gliosis in the right posterior horn seems to be quite isolated.

This case therefore resembles Case 1 in showing, first, that there is a direct transition from a distinct tumour-like mass to a process of gliosis, and secondly that the morbid process terminates below, not around the central canal, but in the region of the posterior columns.

None of the cavities show any trace of an endothelial lining, and they nowhere communicate with the central canal. The new growth appears to have undergone various forms of degeneration, all of which no doubt tend to cavity formation. At some parts there is a fatty metamorphosis, as shown by osmic acid; at other parts there is a myxomatous change, and this is specially seen in the walls of the vessels, many of which are very thick, and appear remarkably homogeneous. The enormous development of vessels in and around the new growth is one of the most striking features: the abnormal state of the vessels—the thick, homogeneous walls and the narrow or obliterated lumen—especially towards the centre of the growth, must lead readily to cavity formation, by softening from malnutrition and absorption of the *débris*. The

cavities seen in fig. 8 have almost certainly an origin of this nature; and the difference between the irregular, broken-down walls of the cavities on the left side, and the comparatively sharp cut margins of those on the right side is probably a difference in the stage of absorption of the broken-down *débris*. The presence of a fibrin-like material in some of the cavities, suggests that hæmorrhage assists in the formation of these spaces. The condition of the central canal varies: at the levels from which figs. 6 and 7 were taken there is no trace of it, while at the level of fig. 8 it presents an appearance as typically normal as we have ever seen; *i.e.*, it is lined by a single, uninterrupted layer of cylindrical epithelial cells, it is not larger than usual, and there are only a few nuclei around it. On the other hand, many sections taken from the dorsal or lumbar region show a central canal larger than usual, but still probably within the normal limit, with the single layer of cylindrical lining cells interrupted at some parts, and a slight proliferation of small cells around it. Comparison with the central canals of a large series of cords, normal and morbid, in which there was no suspicion of such a condition as hydromyelia or syringomyelia, has led us to the conclusion that there is no part of the central canal of Case 2 which can be called undoubtedly abnormal: it is very difficult, however, to lay down the limits of a normal central canal.

With regard to the nature of the new growth, we believe it is the result of the proliferation, not only of the neuroglia, but also of the true nervous elements. The large bodies (fig. 9), which were described as occurring in the new growth, about the level of the sixth and seventh cervical nerves, must, we think, be of the nature of ganglion cells, and not neuroglia cells, for the following reasons:—some are as large as the ganglion cells of the anterior horns, and have large nuclei; there is an entire absence of the fibrillary network of processes so characteristic of neuroglia cells; they occur in greatest numbers in the region of the intermediate grey substance; and their method of staining is identical with that of ganglion cells. They do not, however, show the nucleolus usually so distinct in ganglion cells, nor do they

contain pigment granules. There are also a few nerve-fibres, rather varicose or moniliform in appearance, scattered throughout the growth at this level. We believe, then, that this is an example of that rare form of tumour, known as *neuroglioma ganglionare* or *cellulare*, a name first suggested in connection with a case reported by Baumann.[2] The description and figure given by Ziegler[3] correspond very closely to the condition in our case. In the case recorded by Baumann, there were two tumours of the cerebrum, of which the most striking features were large cells, possessing the characters of ganglion cells, and an abnormal number and arrangement of nerve-fibres: this is, however, rather a case of heterotopia of the grey matter than a true neuroglioma ganglionare. The only examples of the latter, which we can find recorded, are those of Klebs,[4] who describes neurogliomata of the cerebrum, cord, and peripheral nerves; two cases of neurogliomata of the cord are recorded at pages 75 and 77, the most conspicuous features being large ganglion cell-like bodies, and broad bands possessing the character of hyperplastic nerve-fibres. Although we cannot agree with his conclusion as to gliomata in general, viz., that they "arise *mainly* from the true nervous elements of the central nervous system," we believe that a true neuroglioma ganglionare may occur, just as a true neuroma may.

We have seen that in the upper and mid-dorsal regions carmine-stained sections showed homogeneous bodies, and sections stained with osmic acid, showed black masses and particles, especially in the region of the central grey matter (fig. 10). These appear to be due to the same phenomenon, viz., a fatty degeneration of the nervous elements, and this is in accordance with the fact noted by the Weigert-Pal method of staining, that there is a great deficiency of the normal fibrillary nerve plexus in the grey matter of this region.

[2] Baumann. "Beiträge v. Zeigler," II., 1888, s. 500-508.

[3] Ziegler. "Lehrbuch der allgemeinen Pathologie und der pathologischen Anatomie," 8. Auflage, Bd. I., s. 417-418.

[4] Klebs. "Präger, Vierteljahrschr. f. prakt. Heilkunde," 1877, s. 1.

CASE III.—*Left hemiparesis; right leg weak; no wasting of muscles; analgesia and thermo-anæsthesia of nose and ears; loss of all forms of sensation on right half of body (except face); patchy dissociation of touch from pain and temperature on left half of body—left arm and left leg below knee unaffected; sweating only on right side; small recurring growths on ring fingers; cervical spine concave to right. Death within four years of onset. New growth and cavity formation in cord implicating the ventral part of both posterior columns and the left posterior horn; marked degeneration of crossed pyramidal tract and antero-lateral descending tract of left side.*

T. H., aged 25, a coalporter, was admitted into King's College Hospital, under Dr. Ferrier, on July 24, 1892, complaining of difficulty in walking and weakness of the left arm. The following is an abstract of notes taken at that time and during the subsequent course of his illness:—

The family history shows nothing worthy of note.

Patient never suffered from any illness before the onset of the present symptoms. At the age of 5, he was knocked down by a cart, the wheel passing over the left shoulder and arm without causing a fracture.

History of present illness.—About July, 1889, when going to work one morning he suddenly felt his left arm "go to sleep," the numbness commencing in the hand. As this numbness continued for several weeks he was admitted into the West London Hospital, and underwent an operation there on the supposition that "something had formed under the biceps"; this gave no relief. After this he resumed work, but about a year later he suddenly lost almost entire power of the left arm. Under faradic massage, the arm regained strength sufficient to enable him to resume work. About three months later, he lost power in the left leg, the paresis coming on rapidly. His right leg also became weak, and it was found at this time that he had lost almost entire sensibility to touch and pain on the whole right half of the body, except the face. On one occasion he tore open his right hand very badly without feeling it.

Condition on admission is noted as follows:—Patient is a healthy-looking, well-nourished man. He stands with his shoulders and head bent forwards, and stoops towards the right side owing to a slight lateral curvature of the spine with concavity towards the right, the left shoulder being distinctly higher than the right. The chin droops on the chest from weakness of the extensors of the neck. There is marked weakness of the

muscles of the left shoulder girdle, and paresis of both legs. He cannot dorsally flex the left ankle. He walks with left leg very stiff, and dragging his left foot on the ground. He cannot toe the line or walk steadily with eyes shut. All muscles react readily to faradism. The left trapezius and biceps react less strongly, but the left deltoid rather more strongly than the cor-

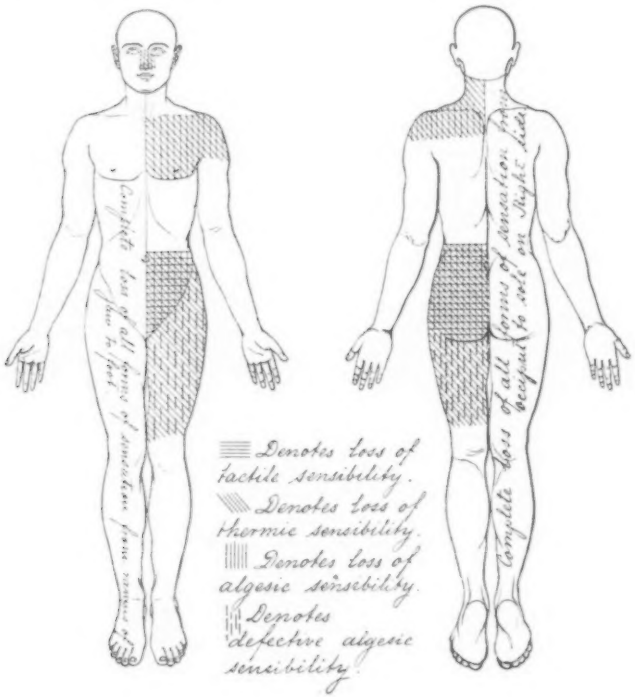

Denotes loss of tactile sensibility.
Denotes loss of thermic sensibility.
Denotes loss of algesic sensibility.
Denotes defective algesic sensibility.

CHART 3.

responding muscles on the right side. The right knee-jerk is active, the left very exaggerated; there is well-marked left ankle-clonus, but only a slight tendency to right ankle-clonus. The plantar reflexes are normal. There is sometimes considerable difficulty in micturition, but never incontinence. Constipation is very obstinate.

CONDITION OF SENSATION.—*Subjectively*, a burning sensation of the sole of the right foot is very persistent. *Objectively*, *see Chart* 3.

On the head and face, sensation of all forms is quite normal, except that there is complete loss of thermic and algesic sensibility with retention of tactile sensibility over the nose and ears.

On the right half of the neck, trunk and right limbs, there is complete loss of all forms of sensibility, including loss of muscular sense, and sense of position in the right arm.

On the left half of the body the affection of sensation is more complicated. Thus, *on the neck*, there is loss of thermic sensibility posteriorly, but not anteriorly. *On the trunk*, there is loss of thermic and algesic sensibility over the shoulder, anteriorly and posteriorly, and over the front of the chest from the clavicle to a little below the nipple. There is loss of all forms of sensibility over the whole lower part of the trunk anteriorly and posteriorly, below the level of a line lying a little above the umbilicus. Over the rest of the trunk, and over the left side of the genitals, sensibility of all forms is normal.

On the left arm sensation is unaffected; while on *the left leg* there is loss of thermic and diminution of algesic sensibility over the thigh and leg as far down as the upper border of the patella anteriorly, and the upper extremity of the popliteal space posteriorly. The rest of the leg is normal.

Trophic Effects.—There is no obvious wasting of muscles anywhere, no cyanosis of the skin, no hyperidrosis. On exertion or after injection of pilocarpine, only the right half of the body perspires. On the exterior surface of the proximal phalangeal articulation of the left ring finger, and on the interior surface of the distal phalangeal articulation of the right ring finger are small recurring growths. Each commences as a small roughness of the skin, which increases till it reaches about the size of a sixpence. Generally it cracks at some period of its growth so that a hard, dry, rough elevation is formed, which gradually heals entirely, but recurs later in exactly the same spot. No similar marks have been noted elsewhere.

The spine shows no prominence of, or tenderness over, any part. The function of all cranial nerves is normal. The right pupil is larger than the left. Lungs, heart, digestive and urinary systems are normal.

September 15.—Patient burnt two large blisters on the outside of the right forearm without feeling any pain.

November 15.—Patient's general condition remains unchanged. Extension of the head is now very feeble, and all movements of the left arm are very weak, especially abduction of the shoulder.

The right arm shows no loss of power, but when the arms are extended, athetoid movements are observed in the fingers of the right hand; while they occur very slightly, and only occasionally in the fingers of the left hand. All the muscles of the arm react to faradism, but those of the right arm rather more readily than those of the left. The left leg has become weaker and is dragged more in walking. The knee-jerks are much exaggerated and

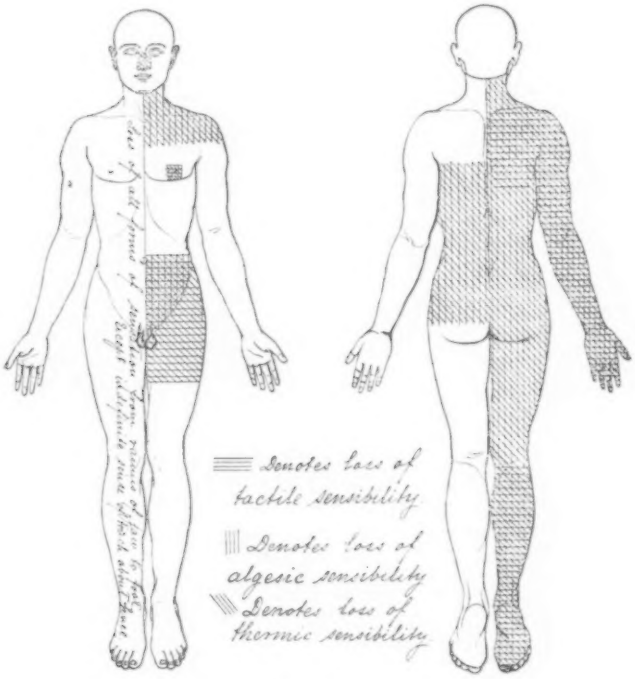


CHART 4.

equal. There is double ankle-clonus, perhaps rather more marked on the left side. The right plantar reflex is greater than the left. The right pupil is still larger than the left; otherwise, the condition of the pupil and ocular movements are normal. The left side of the face acts rather more strongly than the right, and the tongue is protruded slightly to the right. Very obstinate constipation and occasional difficulty in micturition (but never

incontinence) are still present. Swallowing is quite normal. There is no tenderness over the spine.

The *condition of sensation* on this date is given on Chart No. 4.

Head and Face.—Sensibility remains as before, *i.e.*, normal, except for loss of thermic and algesic sensibility over the nose and ears.

Right Side of Body.—*Anteriorly*, there is loss of all kinds of sensibility over the whole of the right half of the body below the ramus of the jaw, except that at or about the knee there is an indefinite sense of touch.

Posteriorly, there is loss of algesic and thermic sensibility all over, from occiput to sole, and loss of tactile sensibility over the whole of the arm, the scapular region, and the leg beneath the knee. Elsewhere, tactile sensibility is normal.

There is complete loss of muscular sense and sense of position in the right arm.

Left Side of Body.—*Anteriorly*, there is loss of algesic and thermic sensibility over the neck, shoulder, and chest down to midway between the clavicle and nipple. From this level to the umbilicus, sensibility is unaffected, except over a small area around the nipple, which has lost all forms of sensibility.

The abdomen, below the umbilicus, has lost all forms of sensibility, but the genitals are unaffected.

The upper half of the thigh has lost tactile and thermic sensibility; the rest of the leg is unaffected.

Posteriorly, tactile sensibility is normal all over, but there is loss of algesic and thermic sensibility from a little above the lower angle of the scapula down to the gluteal region.

January 7, 1893.—Radical cure of hernia performed to-day. Patient began to be troubled with a cough; his breathing got worse, and he died on January 10.

The autopsy showed nothing abnormal in the lungs or anywhere else, except in the spinal cord.

The *microscopical examination* of the cord was imperfectly performed, owing to a breakdown in the method of hardening. At the level of the fourth cervical nerves (fig. 11), the anterior parts of both posterior columns are occupied by an irregularly-shaped cavity, surrounded by a neuroglia-like tissue. There is no evidence of a definite lining membrane to the cavity. The neuroglial tissue is seen to pass posteriorly as a finger-like process, between the columns of Goll and Burdach on either side, both these columns showing secondary degeneration, especially Goll's column.

The new tissue reaches the neck of both posterior horns, and in the left posterior horn there lies a cavity surrounded by neuroglial tissue, this cavity being continuous with the larger cavity in the posterior columns already mentioned. There is no appearance of a central canal, but the anterior commissure is intact. The vessels inside the cord, and in the pia-arachnoid, are large, congested, and distended with red blood corpuscles.

Besides the degeneration already noted in the posterior columns, there is almost complete degeneration of the left crossed pyramidal tract; and the degeneration is continued forward in the position of the antero-lateral descending tract, as far as the antero-median fissure. There is no degeneration in the antero-lateral region of the right side.

At the level of the twelfth dorsal nerves (fig. 12) there is seen to be a well-marked central canal, and from the posterior surface of the grey commissure there extends backwards a wedge-shaped area of neuroglia-like tissue into the postero-median septum. In some of the sections at this level, a few normally stained nerve-fibres are observed cutting off the central-canal region from the wedge-shaped area of new growth. The cavity in the left posterior horn still persists, but it is now quite cut off from the region of the central canal; it is surrounded by a fair amount of neuroglia-like tissue. At this level, there exists the same marked degeneration of the left crossed pyramidal tract, extending forward to a slighter extent into the antero-lateral region.

Remarks.

In this case, as in Cases 1 and 2, the new growth and cavity formation involve the grey commissure, the intermediate grey matter and the anterior parts of both posterior columns; but here only one posterior horn—viz., the left—is implicated, and the secondary descending degeneration is limited to the left side. This preponderance of pathological affection of the left side accords with the clinical facts that the paresis was almost limited to the limbs of the left side, there being only slight weakness of the right leg, while the affection of all forms of sensation was greatest on the right side, and the sense of touch was implicated almost exclusively on that side, *i.e.*, clinically it is an example of Brown-Séquard's palsy.

In the midcervical region, the communication of the

cavity in the posterior horn with the cavity in the posterior columns is very distinct, and there is certainly no trace of an endothelial lining to any part of the cavity.

The cavity in the left posterior horn in the lower dorsal region is probably continuous with the cavity in the corresponding situation in the midcervical region, but the absence of a continuous series of sections renders this uncertain.

The extension of the new-growth in the midcervical region along the septum between Goll's and Burdach's column on either side, and the limitation of the gliosis in the lower dorsal region to a wedge-shaped area in the front part of the postero-median fissure are examples of implication of two favourite sites for syringomyelic changes (Schlesinger[5]).

Some General Remarks on the Conduction of Sensory Impressions in the Spinal Cord.

It has been assumed in many cases that the sensory symptoms of syringomyelia are due to the interruption of the afferent conducting tracts in the spinal cord; but, as Dr. Ferrier pointed out, when the above recorded cases were exhibited before the Neurological Society, the anæsthesia was mainly segmental in distribution, sensation not being affected in the parts below the lesion—except in one instance (Case 3).

Hence these cases show that the pathological changes occurring in syringomyelia may produce disturbances of sensation in one of two ways:—

(*a*) By interfering with incoming sensory impressions through one or more posterior roots—segmental anæsthesia.

(*b*) By severing the continuity of the conducting paths, thus abolishing sensation in the parts of the body below the lesion.

We find examples of both conditions in the cases here recorded. Thus, in Cases 1 and 2, throughout the whole course of the disease, the defects of sensation were limited to the spinal segments corresponding roughly to the pectoral

[5] *Schlesinger*, "Die Syringomyelie," 1895, pp. 159-162, and fig. 14.

girdle, and illustrating the segmental nature of the anæsthesia (*vide* charts 1 and 2). In Case 3 both varieties of sensory disturbance were observed; on one side a segmental anæsthesia, corresponding to certain posterior nerve-roots; on the other, total anæsthesia below the lesion from interference with the afferent paths (*vide* charts 3 and 4).

This condition is what might be expected from the *post-mortem* examination of the spinal cords, already detailed and figured. Here we note that two individual parts of this structure are seats of election of new growths:—

(*a*) The posterior root-zones, and adjacent parts of the postero-median columns.

(*b*) The gelatinous substance of Rolando and adjacent parts of the posterior horn.

Interference with the former structures, more especially the posterior root-zone, is in all likelihood, capable of effecting the segmentary sensory disturbances; while the anæsthesia below the seat of the lesion is due, in our opinion, to destruction of the posterior horn and gelatinous substance of Rolando. Case 3 is highly instructive in that both conditions were observed. On one side—the right—the new growth is limited to the anterior portions of the posterior root-zone and adjacent part of the postero-median column (fig. 11). The sensory defects arising from this lesion are segmental in nature, and are upon the same side of the body, but, as shown in charts 3 and 4, they are included in and obscured by the anæsthesia from lesion of the conducting tracts on the opposite side. On the other side—the left—there was a lesion involving the posterior horn and gelatinous substance as well as the posterior root-zone and adjacent part of the postero-median column, in the upper cervical region of the spinal cord, as a result of which all forms of sensation were abolished on the right side below this level; while there was segmental anæsthesia on the left side of the body (charts 3 and 4). As there existed on the side of the lesion much weakness of the arm and leg, with descending secondary degeneration in the pyramidal tracts, there was presented the classical symptomatology of the so-called Brown-Séquard paralysis. In a previous number of

this Journal (BRAIN, pts. 70 & 71, p. 231) it was shown that experimental destruction of the tubercle of Rolando and the adjacent part of the posterior horn in monkeys was productive of a constant series of objective sensory phenomena, consisting mainly of abolition of all forms of sensation over the area of trigeminal distribution on the side of the lesion, and loss of response to painful impressions on the opposite side, the response to ordinary tactile impressions being temporarily abolished on the side of the lesion. The conclusion drawn from these experiments was, that the gelatinous and spongy portions of the posterior horns were of prime importance in the conduction of sensory impressions at all events in the upper portions of the spinal cord, more especially in the neighbourhood of the spinal-medullary junction.

The large number of experimental observations made within recent years upon hemisection of the spinal cord by Mott, Marshall,[6] and others, tend to show that there is no constant distribution of the sensory fibres, which enter by the posterior nerve roots. The cases of unilateral lesion of the spinal cord in man, on the other hand, show a much more constant relation between the several forms of cutaneous sensibility. Reference to a large number of cases of the so-called Brown-Séquard paralysis, scattered throughout medical literature, has shown that the sense of pain is invariably abolished on the side opposite the lesion in association with the sense of temperature, while these are not uncommonly dissociated from tactile sensibility. Whether the sense of touch is abolished along with the other forms of cutaneous sensation, or not, would appear to depend upon the extent of destruction of the conducting paths, as is presently to be shown.

And this leads us to the consideration of one of the most interesting features brought out by a study of these cases, viz., the curious dissociation which exists between the several forms of cutaneous sensation. In Cases 1 and 2 loss of thermic sensibility was first observed. In Case 1 there

[6] *Proc. Roy. Soc.*, 1895, p. 475.

existed throughout the whole course of the disease a dissociation between thermic sensibility on the one hand, and tactile and painful sensibilities on the other. But in Case 2 there occurred a progressive interference, with eventual abolition, of the sensibility, first of temperature, then of pain, and lastly of touch.

These facts do not necessarily indicate the existence of separate tracts in the spinal cord for the conduction of the several forms of cutaneous sensation. As the order in which they are lost appears to be constant, it is difficult to believe that disease should invariably at the outset implicate tracts subserving thermic sensibility, and later on those of pain and touch. This may be explained on the hypothesis *that the more fundamental the form of sensation, the more extensive is the provision for its conduction in the spinal cord.*

Hence in the early stages, when the lesion is small, thermic sensibility only is impaired; while in the late stages, when the destruction of tissue is presumably greater, the less organised forms of sensation are impaired or abolished; and, conversely, after a spinal lesion, which has at the outset abolished all forms of sensation, the appreciation of touch is that which first returns, while pain and temperature usually remain in abeyance. In amplification of this we have seen a monkey, in which both painful and tactile sensibilities were abolished by a lesion involving the posterior region of the spinal cord. Although the loss of pain persisted, the sense of touch returned on the side which was least involved, a circumstance which may find explanation in the more widespread distribution throughout the posterior horn of the fibres subserving tactile sensibility. (Brain, *loc. cit.*, p. 238).

To look more particularly at the individual cases, it is clear that this generalisation holds good, whether the lesion involves the conducting or the segmental arrangements. Thus, in cases where the posterior root zones were only or chiefly implicated, loss of thermic sensibility was that first affected, and in Case 1 this was the only form of cutaneous sensibility abolished.

This view harmonises with what must undoubtedly occur,

both in the peripheral nerves and in the end-organs in the skin. It is as difficult to realise the existence of separate end-organs, or of separate nerve fibres,[7] for the reception and conduction of the several forms of cutaneous impressions, as it is to assume separate paths for their transmission in the spinal cord, or separate centres for their perception in the cerebral cortex.

EXPLANATION OF THE PLATES.

Figs. 1—5 represent the appearance of the Spinal Cord in Case I.

FIG. 1.—Mid-cervical region.
FIG. 2.—Lower cervical region.
FIG. 3.—Upper thoracic region.
FIG. 4.—Lower thoracic region.
FIG. 5.—Lumbar enlargement.

Figs. 6—10 are taken from Case II.

FIG. 6.—At level of second and third cervical nerves.
FIG. 7.—At level of fifth cervical nerves.
FIG. 8.—At level of sixth and seventh nerves.
FIG. 9.—Neuroglioma ganglionare (Eosin and Logwood).
FIG. 10.—Central grey matter (Marchi's method).

Figs. 11 and 12 depict the Spinal Cord of Case III.

FIG. 11.—At the level of the fourth cervical nerves.
FIG. 12.—At the level of the twelfth dorsal nerves.
Detailed descriptions of the figures are given in the text.

[7] In some interesting experiments by Waller, thermic sensibility was the last to return after compression of the trunk of the radial nerve (referred to by Mitchell. "Remote consequences of injuries to nerves," 1895, p. 21).

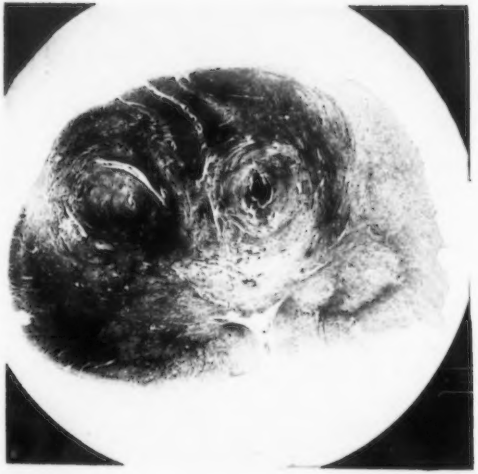


Fig. 1.

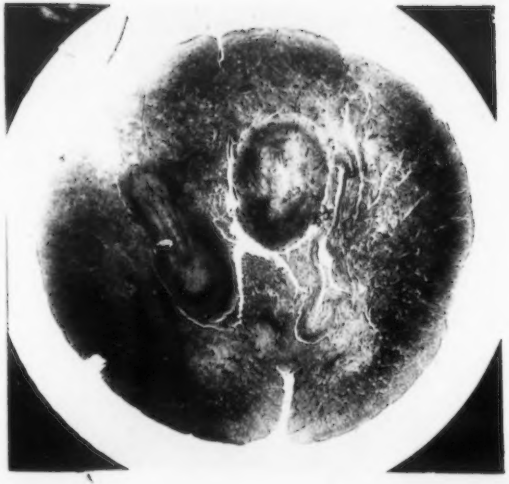


Fig. 2.

Collotype. J. J. Briginshaw, Sise Lane, 35d, Queen Victoria Street, E.C.

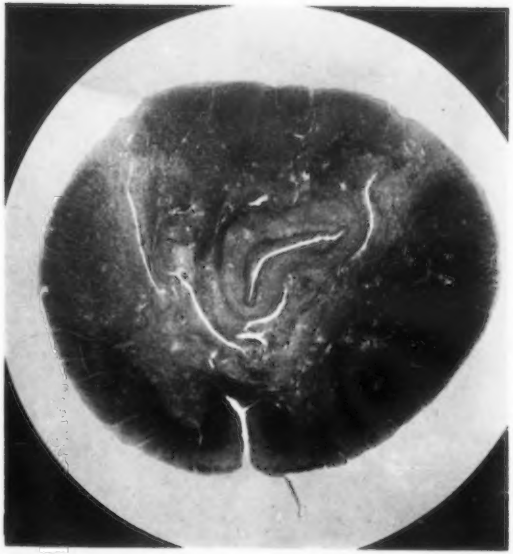


Fig. 3.

Fig. 4.

Collotype J. J. Briginshaw, Size Lane, 35d, Queen Victoria Street, E.C.

Fig. 5.

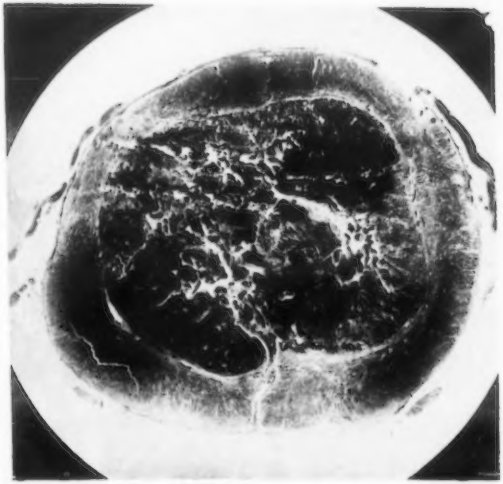


Fig. 6.

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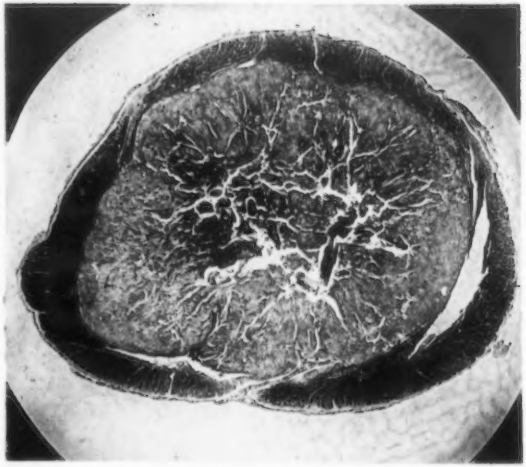

Fig. 7.

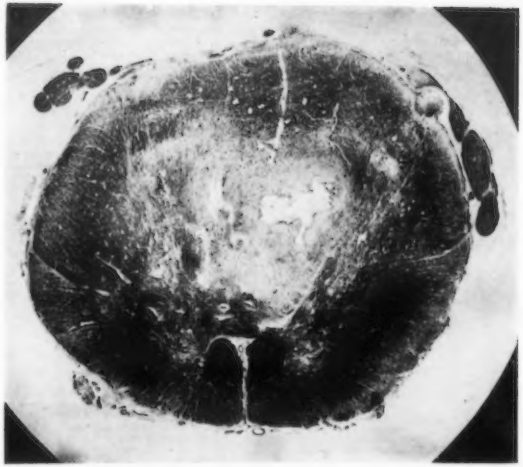

Fig. 8.

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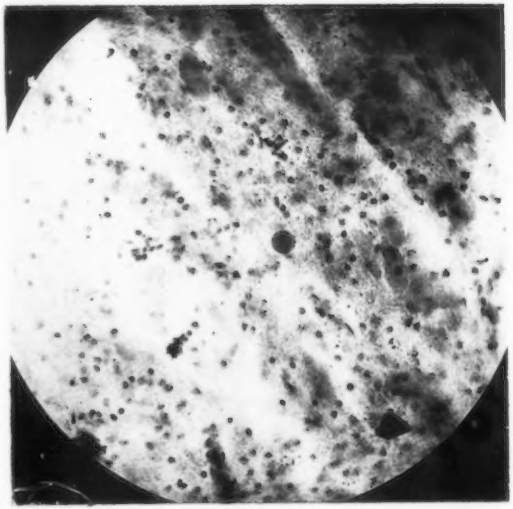

Fig. 9.

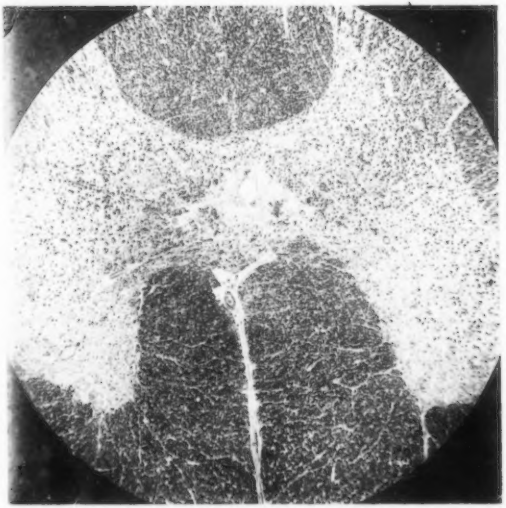

Fig. 10.

Collotype. J. J. Briginshaw, Sise Lane, 35d, Queen Victoria Street, E.C.

Fig. 11.

Fig 12.

Collotype. J. J. Briginshaw, Sise Lane, 35d, Queen Victoria Street, E.C.

ON A DESCENDING DEGENERATION IN THE POSTERIOR COLUMNS IN THE LUMBO-SACRAL REGION OF THE SPINAL CORD.

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In 1867, Bastian (1) figured a descending tract in the posterior columns of the cord, which had degenerated subsequently to an injury in the upper part of the cervical enlargement.

Thirteen years later (1880), Kahler (1) and Pick (2) described a somewhat diffuse descending degeneration in the posterior columns after fracture of the sixth cervical vertebra. In the same year, Strümpell (3) described and figured a small area of descending degeneration in the dorsal region, following a transverse myelitis. This area was situated at the junction of the postero-median and postero-external columns, near the posterior commissure. Westphal (4) also figured and described a downward degeneration of "two narrow bands parallel to the posterior horns" as being present at the ninth dorsal root, in a case in which there was a tumour at the fourth and fifth dorsal segments. His figure shows the degenerated area to be in the postero-lateral column, but not quite reaching either the posterior commissure or the periphery of the cord.

In 1883, Schultze (5), in a case of tumour at the level of the fourth and fifth cervical roots, found a degenerated tract in each postero-external column, which descended for about $2\frac{1}{2}$ centimetres (nearly one inch). He described and figured it as beginning some distance from the posterior commissure, and terminating some little way from the periphery, and as being parallel to the inner margin of the posterior cornu. He termed it, from its form, the "comma-shaped" tract, which name it still retains. It is quite evident that this tract is the same as that described by Westphal. Schultze regards it as being formed of descending branches of the posterior root fibres, but without adducing any adequate reason for his opinion.

Tooth (6), in 1889, described three cases in which this tract was affected. He found that the degeneration of the comma-shaped tract extended as low as the eighth dorsal segment, in a case in which the cord was crushed between the eighth cervical and first dorsal roots; in another case, in which the cord was crushed between the sixth and seven cervical roots, the degeneration extended to the sixth dorsal segment; and, in the third case, a fracture dislocation of the fifth dorsal vertebra, the degeneration extended an inch below the lesion. He also found that the tract degenerated downwards on the same side, after hemi-section of the cord, and considers that the degeneration is probably more distinct after lesions above the fifth dorsal root than below that level. He failed to find any degeneration in this tract after section of the roots only, and, therefore, regards it as being probably commissural in nature.

Schmaus (7), in 1890, described and figured an area of descending degeneration in the lower cervical and upper dorsal cord, which closely corresponds in form and position with the comma-shaped tract, as described by Schultze and Tooth. The degeneration was due to a softening in the cervical enlargement, following an injury resulting from a fall.

Pfeiffer (8), in 1891, in a case of sarcoma, involving the three lower cervical and first dorsal segments, found at the level of the middle and lower dorsal roots, a degeneration involving part of the postero-external, and the adjacent portion

of the postero-median columns. In the absence of figures one cannot form any more precise idea of the position and shape of the degenerated tract in this case. It did not, however, extend below the lowest dorsal segment.

Bruns (9), in 1883, figures a descending tract in the postero-external columns, which degenerated as far as the fifth dorsal segment, after a lesion at the junction of the cervical and dorsal regions. He terms it the "comma-tract," but his figures show a somewhat more extensive degeneration than is indicated by Westphal, Schultze, and Tooth.

Gombault and Philippe (10), in a case of gradual compression by a tumour, at the third cervical segment, found a descending degeneration in a narrow band in the posterior columns, situated nearer Goll's column than the posterior horn, but not reaching either the commissure, or the periphery. It extended downward nearly to the sixth cervical segment. They agree with Tooth in thinking that the "comma tract of Schultze" is not directly connected with the descending fibres of the posterior nerve-roots, since they found it undegenerated in a case (of lead poisoning) in which the fifth cervical root was destroyed.

These cases have established beyond doubt (*a*) that in the cervical and dorsal portions of the cord, as low down at least as the ninth dorsal segment, there is in the postero-external column a tract, which degenerates in a descending direction; (*b*) that this tract is situated in the position first described by Schultze, namely, about the middle of the breadth of the postero-external column, parallel to the inner margin of the post cornu, and not quite reaching either the posterior commissure or the periphery of the cord; (*c*) that the degeneration of this tract passes downwards for only a limited distance. It is as yet uncertain what this distance is, how the tract begins, or how it terminates. Further research is required to settle these points; but it seems clear that the tract is not found in the lumbar region in the same relation to the posterior cornu, and that, if it exists there at all, it must take the form of a diffuse degeneration, or occupy some other position. None of the above-mentioned observers, however, with the

exception of Gombault and Philippe, appear to have found any degeneration in this region. Daxenberger (12) describes a descending degeneration, extending from the first dorsal segment, as far downward as the upper lumbar region, in both the postero-external and postero-median columns. The degeneration in the former was comma-shaped, and was present in the upper and middle dorsal regions. In the postero-median column, the degeneration lay close along each side of the posterior median septum, in its anterior half, but not reaching quite to the commissure; and was present throughout the dorsal and upper lumbar regions. This degeneration in the postero-median column has not been found in any other case, and it seems difficult to reconcile it with the facts already mentioned, or with the fact that ascending degeneration of this column leaves no unaffected area, as it ought to do, if the tract in the cervical and dorsal regions contained descending fibres.

We have recently, however, had an opportunity of examining a case which appears to us to establish definitely the existence of a descending tract in this region. In a case of fracture-dislocation of the twelfth dorsal vertebra, the cord was completely crushed and destroyed in the upper lumbar region, over a length of about one inch, death taking place within five weeks of the injury—at a time, therefore, specially favourable for ascertaining the degeneration by Marchi's method. The cord was hardened for four weeks, in the usual way, in Müller's fluid, and then small pieces at various levels, above and below the lesion, were transferred for fourteen days to Marchi's fluid.

In sections made from the lumbar and sacral regions, a diffuse degeneration was found throughout the whole of the antero-lateral columns. This degeneration was fairly uniformly distributed, except in the position of the crossed pyramidal tracts, where it was rather more marked, and in the anterior columns, along the margins of the anterior median fissure, and the adjacent portion of the anterior surface of the cord (the *descending sulco-marginal tract of Marie*). These degenerations diminished in a descending direction.

In the posterior columns there were various degenerations. Immediately below the spot at which the cord had been destroyed, these columns were degenerated in an irregular manner, the degeneration being manifestly dependent upon the direct injury to the fibres. It should further be noted that several of the posterior roots in the upper lumbar region, not extending below the fourth lumbar segment, had been injured between the cord and the posterior root ganglion, but that the roots below this level were quite intact.

At the level of the third lumbar segment there was a somewhat dense degeneration in the anterior root zone of Flechsig. This was undoubtedly, for the most part, due to the degenerated fibres from the posterior roots, which could be seen entering the zone from behind and leaving it in front, to pass into the grey matter.

A second area of degeneration was found on either side closely applied to the hinder third of the posterior-median septum, and along the inner half of the periphery of the posterior columns. The breadth of these areas, as is shown in the diagram (fig. 1), was somewhat greater along the periphery than along the posterior median septum, where, on the other hand, the degenerated fibres were more closely aggregated.

Thirdly, there was a slight diffuse degeneration in the middle root zone. The part of the posterior columns which lay next the middle third of the septum, between the periphery and the posterior commissure, shewed almost no degeneration.

At the level of the fourth lumbar segment (fig. 2.) the degeneration along the inner margin of the posterior cornua had become much less marked, evidently because at this level the degenerated root fibres of the injured nerves had almost entirely ceased to enter the cord. The second area of degeneration, namely, that along the posterior median septum and the adjacent portion of the periphery of the cord, had greatly increased in extent. There was now a narrow band of degenerated fibres, extending along the whole length of the septum, from the posterior commissure to the

the periphery. The fibres were more closely aggregated, and the band was rather thicker at the posterior portion of the septum than in the anterior two-thirds. On the other hand, the degeneration at the periphery did not extend quite so far in a lateral direction as at a higher level. In the middle root zone there were practically no degenerated fibres.

In the upper sacral regions (figs. 3 and 4), the median degeneration had assumed a distinctly triangular form posteriorly, while still extending forward as a narrow band on either side of the septum as far as the posterior commissure, and apparently extending into the grey matter. At the junction of the lumbar and sacral segments the degenerated area had apparently attained its maximum. In the lower sacral and coccygeal areas it gradually diminished in size, while retaining the same defined wedge-shape (figs. 5 and 8). In the three lower sections figured the degenerated fibres could be traced into the bases of the posterior cornua at either side of the central canal.

There is no doubt whatever that this degeneration is a descending one. There were no injured roots below the level of the fourth lumbar segment which could possibly have given rise to it, and there was no other injury of the cord at its lower end. It would appear most probable that the tract increases in size by the gradual passage into it of degenerated fibres from other regions of the posterior column at higher levels, but we are unable to determine directly whether the tract is formed from descending fibres of the posterior roots in the lumbar region, or within the cord itself. This point might be settled by cases of section of, or tumours within, the cord on the one hand, or, on the other, by affections of the nerves produced by section or destruction of the cauda equina, or by disease, such as locomotor ataxia, which leads to degeneration of the root fibres within the cord. If the fibres are of endogenous origin, that is, if they originate within the cord, they would degenerate after lesions within the cord, and would be spared in injury or lesion, involving the roots only.

Barbacci (11), in a case of neoplasm in the sixth dorsal segment, found a descending degeneration in the posterior

columns, which at the level of the eighth dorsal segment was diffused over all the areas, but was most marked in the portion of the postero-external column next the posterior cornu. At the level of the last dorsal and first lumbar segments, this area tended to become grouped more towards the inner part of the postero-external column, and at a still lower level appeared as a narrow band along the posterior half of the posterior median septum. This case is referred to by Marie ("Lectures on Diseases of the Spinal Cord," Sydenham Society's translation, p. 38). Barbacci also found that, after section of the cord of the cat at the second and third lumbar segments, there were a few degenerated fibres diffusely scattered throughout the whole posterior column. In another case, after hemi-section of the cord of a dog between the tenth and eleventh dorsal roots, there was at the lower dorsal region, a descending degeneration in the posterior columns which formed an irregularly triangular area near the hinder part of the septum. There were also some degenerated fibres scattered throughout the rest of the column. At the level of the lumbar enlargement only a few degenerated fibres remained, situated mostly in the anterior portion of the column.

Gombault and Philippe (*loc. cit.*) in a case of total transverse myelitis in the upper part of the lumbar enlargement, found below the lesion, on either side of the posterior median septum, a triangular area of degeneration, which on the right side almost reached to the posterior commissure. At lower levels this triangle receded gradually from the posterior commissure, and ultimately its apex lay midway between the commissure and the periphery of the cord.

On the other hand, in two cases described by Schultze, in one of which the cauda equina had been crushed, and in the other the roots of the sciatic nerve and the lumbar portion of the cord had been destroyed, a small triangle of undegenerated fibres was found above the lesion at the junction of the posterior median septum with the periphery of the cord. The rest of the posterior columns, with the exception of the cornu-commissural tract of Marie, was degenerated.

Gombault and Philippe further describe a case of loco-

motor ataxia in which the fourth and fifth sacral roots on both sides, and the sixth sacral (coccygeal?) root on the right side, were destroyed. Here they found a tract of undegenerated fibres in relation to the posterior median septum. This tract, at the level of the fifth and sixth sacral roots, was small and of a triangular form. Above this level it passed further forwards along the posterior median septum, assuming, at the same time, an oval form. At the level of the fourth lumbar it became reduced in size. The authors give satisfactory reasons for concluding that these fibres are not derived from the coccygeal root, but must belong to a descending tract of the cord.

The persistence of similar areas of healthy fibres in the lower lumbar and sacral regions in locomotor ataxia is well known; but the reasons given why they fail to degenerate along with the adjacent portions of the posterior columns have hitherto been unsatisfactory. If they represent, as we now believe they do, fibres descending from higher levels of the cord, it is quite intelligible why they have escaped the degeneration which has overtaken those fibres which are a continuation of the posterior roots within the cord.

Hoche (13) describes two cases of compression of the cord, one at the eighth cervical, and the other between the fourth and sixth dorsal roots, of three and six weeks standing respectively, in which the degenerations in the posterior columns in the dorsal, lumbar, and sacral regions presented closely similar appearances. He found that after a lesion in the cervical region, a descending degeneration could be traced as far as the lowest portion of the conus terminalis. The fibres of the comma-shaped tract ended above the first appearance of the "oval field of Flechsig" (*i.e.*, above the twelfth dorsal root), by passing into the grey matter, and thus took no share in forming the "oval field." In the lumbo-sacral region there was a degenerated area on either side of the posterior median septum, which, in the middle and lower part of the lumbar enlargement, did not quite reach the posterior commissure or the periphery, but in the sacral region extended back to the periphery, and also sent its fibres forward into the grey matter. The lowest fibres of the degen-

eration ended in the conus terminalis by bending forward into the grey substance. This tract, at the level of the twelfth dorsal root, occupied a small area extending along the periphery of the cord from the posterior septum about half way to the entry of the posterior roots. In the upper dorsal region it was further outwards and more diffuse, forming a wedge-shaped area in the postero-external column, with its base at the periphery, and its apex touching the tail of the comma tract. This relation existed as far down as the ninth dorsal segment.

According to Hoche, therefore, the descending tract in the posterior columns in the lumbo-sacral region, and the comma-shaped tract of Schultze, both originate in the cervical part of the cord; but they are quite independent of each other, and the lumbo-sacral tract does not represent a downward continuation of the comma-shaped tract.

Conclusions.

It seems *proved* :—

(1) That there is a descending tract in the posterior columns in the lumbo-sacral region.

(2) That this tract in the upper lumbar region is somewhat irregularly diffused through the greater part of the column.

(3) At the level of the third lumbar root (fig. 1) it becomes collected along the margin of the hinder portion of the posterior-median septum and the periphery of the inner half of the posterior columns.

(4) That at the level of the fourth lumbar root (fig. 2) it extends forward as a narrow band along the side of the septum as far as the posterior commissure. Below this level, at the first and second sacral roots (figs. 3 and 4) the tract has the form of a narrow wedge, with its base at the periphery, and its apex at the posterior commissure. Below the third sacral root (fig. 5) the tract diminishes in size as far as the coccygeal segment (figs. 6, 7 and 8), while retaining its triangular form, and its relation to the posterior median septum.

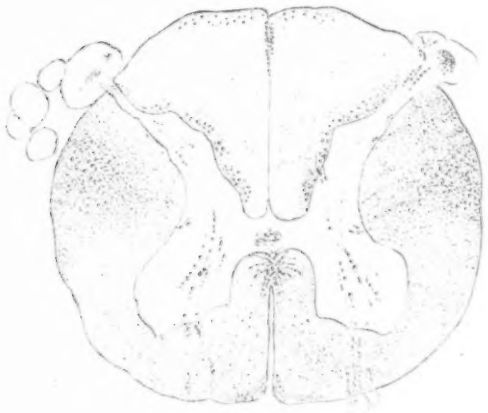

FIG. 1.

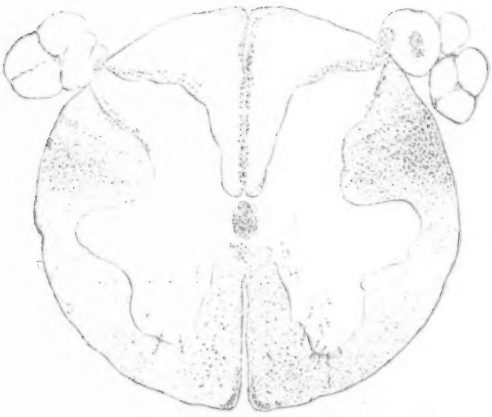

FIG. 2.

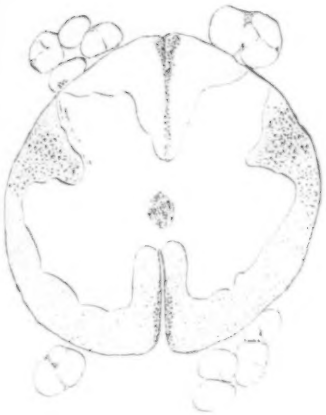

FIG. 3.

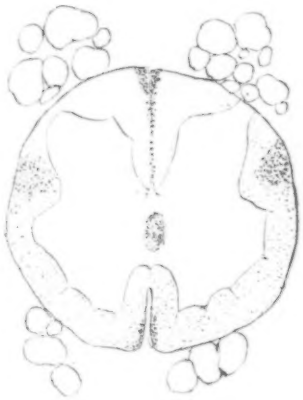

FIG. 4.

FIG. 5.

FIG. 6.

FIG. 7.

FIG. 8.

(5) That its fibres terminate by passing forwards into the grey matter at the base of the posterior horn of the same side (figs. 6, 7 and 8).

It seems *probable*:—

(1) That the tract is not directly continuous with the fibres of the posterior roots in the lumbo-sacral region. This seems indicated by its escape in locomotor ataxia, and in cases of destruction of the cauda equina.

(2) That it is also independent of the "comma-shaped" tract. This seems indicated by the failure of any observer to trace the latter as a distinct tract to a lower level than the ninth dorsal segment.

(3) That it is independent of the cornu-commissural tract of Marie. This tract seems to have entirely escaped degeneration in our case, a fact which seems to indicate that if its fibres have a descending direction, they must originate below the level of the lesion, in the upper lumbar region.

(4) That the fibres which compose this tract probably enter it at different levels.

(5) That it appears to correspond in position to the "oval field" of Flechsig, and the "dorso-median sacral bundle" of Obersteiner (14).

NOTE.—As the tract bears a relation to the *posterior median septum*, analagous to that borne by the *sulco-marginal tract* of Marie to the *anterior median* fissure or sulcus, we would suggest that it may be termed the *descending septo-marginal tract*. This name would indicate briefly its course, its position in the posterior columns, and its relation to the septum.

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EXPLANATION OF PLATES.

Figure 1. T. S. of spinal cord at the level of the third lumbar root.
„ 2. T. S. of spinal cord at the level of the fourth lumbar root.
„ 3 & 4. T. S. of spinal cord at the level of the first and second sacral roots.
„ 5 & 6. T. S. of spinal cord at the level of the third and fourth sacral roots.
„ 7 & 8. T. S. of spinal cord at the level of the fifth sacral and coccygeal roots.

ON THE SYMPTOMATOLOGY OF GROSS LESIONS (TUMOURS AND ABSCESSES) INVOLVING THE PRÆ-FRONTAL REGION OF THE BRAIN.

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THE symptoms produced by tumours and abscesses involving the præ-frontal region of the brain are often somewhat ill-defined, and probably mistakes in localisation occur more frequently in lesions of this region than in lesions of any other part of the brain. In many cases, however, a careful study of all the symptoms will indicate the seat of the disease.

During the last ten years five cases of tumour or abscess involving the præ-frontal region have been admitted into the medical wards of the Manchester Royal Infirmary, and an analysis of the symptoms in these cases, and in forty-five other cases, reported in medical literature during recent years, presents several points of interest.

Experiments on animals have shown that lesions of the præ-frontal region are not followed by such well-defined symptoms as lesions of other parts of the brain. Nevertheless, the results of these experiments are of great interest. They have been ably discussed by Professor L. Bianchi in the last volume of BRAIN (winter part, 1895, p. 497), and hence in this paper need only be briefly referred to. The views which have been advanced as to the functions of the præ-frontal lobe may be summarised as follows (Professor Bianchi):—

"(1) The præ-frontal lobe is the motor centre of the eyes and head on the opposite side, and in consequence of the

close relationship between the movements of these parts and attention, it is also the centre for attention (Ferrier).

"(2) It is the centre for the highest psychical functions. Destruction of it involves a real decadence of psychical activity (Wundt, Hitzig, Bianchi).

"(3) It is part of the so-called 'Fühlsphære,' and as such is the motor centre of the dorsal muscles. Its highest development is not correlated with that of intellect, but with that of the dorsal musculature (Munk, Luciani)."

Professor Bianchi criticises the first and third views and records a number of experiments made on animals. From the psychical disturbance which he has observed after removal of the præ-frontal lobes he concludes, "that the frontal lobes are the seat of co-ordination and fusion of the in-coming and out-going products of the several sensory and motor areas of the cortex." "The frontal lobes would thus sum up into series the products of the sensori-motor regions, as well as the emotive states which accompany all perceptions, the fusion of which constitutes what has been called the *psychical* tone of the individual."

The following are abstracts of the notes of four cases of tumour and one of abscess, involving the præ-frontal lobes. To the kindness of Dr. Leech, Dr. Dreschfeld, Dr. Graham Steell, and Mr. Wright, I am indebted for the opportunity of reporting these cases.

Case 1.—*Sarcoma of both præ-frontal lobes; absent knee jerks; unilateral optic neuritis; spinal cord normal.*—Mary O., aged 23; under the care of Mr. Wright and Dr. Dreschfeld. A sarcomatous tumour had been removed from the middle of the anterior part of the left thigh, but on November 20, 1893, the patient was re-admitted to the Manchester Royal Infirmary, owing to the appearance of a swelling about the size of an egg at the lower part of the cicatrix.

The patient, on admission, complained of a feeling of sickness. On November 23 she still complained of sickness, but had no pain anywhere. On the evening of the 26th she felt very sick and complained of headache on several occasions. On November 27, whilst having a bath, she had what appeared to be an attack of syncope. The pulse became very feeble, and the patient lost consciousness. Ether and brandy were given, but she remained

unconscious. The temperature was 97·6°. On November 28, she still remained unconscious, and the temperature rose to 100·8° in the morning, and 101·6° in the evening. I saw the patient for the first time on November 29. She then appeared to be unconscious. When the arms were raised into the vertical posture and allowed to fall, the right one fell down more suddenly than the left. When the right arm, leg, and right side of the face were pricked with a pin, no movement could be obtained, but slight movements were obtained on the left side. The pin prick appeared to cause pain on both sides. *The knee-jerk was absent on both sides*; plantar reflexes present; no ankle clonus. The abdominal and epigastric reflexes were present on the left side but not on the right. There was slight blurring of the margins of the right optic disc. Heart and lungs normal. November 30 there was slight optic neuritis in the right eye. The left disc was normal. The patient was in a semi-conscious condition, but could be roused by pinching or pricking with a pin. A few days later she would put out the tongue when asked to do so, but the knee-jerks remained absent on both sides. The optic neuritis in the right eye became more marked, but the left optic disc remained normal. On December 12 the difference between the limbs on the right and left sides was very slight. The patient could be made to perform slight movements of the right limbs, though she still remained in a drowsy state. No facial paresis could be detected. The tongue was protruded straight. She could not be made to answer any questions, but would occasionally speak to the nurse. *The knee-jerks were still both absent.* She could swallow food quite well. On December 27 she became quite comatose, and death occurred on the 28th.

The knee-jerks were carefully examined three or four days before death, but *they were both still absent*, and there was no ankle clonus. During the four weeks the patient was in the hospital the knee-jerks were always absent.

At the *post-mortem examination*, on the surface of the left cerebral hemisphere, in the præ-frontal region, was an oval tumour growth about 2 in. by 1¼ in., the long diameter corresponding to the position of the second left frontal convolution. On making vertical transverse sections of the brain, the growth was found to extend deeply (for about 2 inches at one spot) into the white matter of the left præ-frontal lobe. The tumour was very vascular, and at the centre was softened and broken down, giving rise to a cavity which contained yellow fluid and *débris*. In the white matter of the right præ-frontal lobe were two smaller

tumour growths, one just beneath the first frontal convolution, and one beneath the second convolution. The former was softened at the centre; both were vascular. Another section through the posterior part of the third frontal convolution showed the presence of a fourth tumour, situated in the middle of the white matter of the right præ-frontal lobe, and a very small growth invading just the anterior end of the right lateral ventricle.

The ascending frontal convolution, the motor region of the cortex, the motor fibres in the white matter and the basal ganglia were *not* invaded by tumour growth. In the white matter of the occipital lobe on the left side there was a small nodule of new growth about the size of a pea, and one in that of the right occipital lobe almost at the apex of the lobe.

The *cerebellum* medulla and pons were quite normal. The spinal cord was normal to the naked eye; after hardening in Müller's fluid it appeared normal on section. Pieces were taken from the lumbar, dorsal and cervical regions, and embedded in celloid, and sections made. Microscopically the cord appeared normal.

At the inner part of the left thigh, about the apex of Scarpa's triangle, was a tumour growth about the size of an egg. Microscopically, the cerebral tumour was found to be a very vascular sarcoma. There were no changes of importance in the other organs.

Case 2.—Abscess of right frontal lobe. Optic neuritis on right side only. Frontal and occipital headache. Absent knee-jerks.—William B.,[1] aged 19, admitted as an in-patient under the care of Dr. Steell, March 22, 1890. On February 18, 1890, the patient was attacked with influenza. The nasal discharge was profuse, and after recovering from the attack he continued to suffer from pain in the head, especially in the frontal and occipital regions (Previous to the attack of influenza he was not troubled with headache.) His mental condition became very dull and he was unable to return to his work. The pain in the frontal region became so severe that he was obliged to remain in bed. The mental condition became gradually more dull and stupid.

Condition on admission to the Hospital.—Patient is pale and badly nourished. His face has a dull, stupid expression. He takes very little notice of his surroundings, but always recognises his mother. He lies on his back in a drowsy condition, with his

[1] The complete history of this case has been published by the writer in the *Medical Chronicle*, 1890-91, vol. xiii., p. 423.

eyes half closed and his mouth half open. He seldom moves his head. He never speaks unless spoken to, but answers questions quite intelligently and distinctly, though slowly and in a low voice. The patient is constantly buttoning and unbuttoning his shirt, or re-arranging the bedclothes, pulling them a little higher, or turning them down for a few inches. His legs and trunk, however, remain quite still. There is no paralysis of facial muscle, of the tongue, or of the limbs. The movements of the eyeballs upwards and downwards and to the right, are performed quite well, but the patient cannot be made to look to the left. Repeated trials fail to induce him to look in that direction; he always states that he is unable to do so, and the eyeballs remain directed straightforwards. (When examined next day this symptom had disappeared and all the movements of the eyeballs could be performed quite well.) Knee-jerks absent, no ankle clonus, plantar reflexes present. No anæsthesia.

Ophthalmoscopic examination revealed distinct optic neuritis in the right eye, but no optic neuritis in the left eye. The condition remained much the same until March 28, when the evening temperature was 101·2° F. On April 2, the morning temperature was 101°, evening 102·8°; April 3, 103·8° in the morning, 104° in the evening. On April 4, the morning temperature was 105·8°, evening 106·4°. The patient became comatose and death occurred on the evening of April 4.

Post-mortem examination revealed a large abscess in the anterior part of the right frontal lobe. It extended to within $\frac{1}{16}$ of an inch of the surface of the anterior extremity of the frontal lobe, and posteriorly to a point a quarter of an inch in front of the right caudate nucleus; laterally it extended to about a quarter of an inch from the convex surface of the cortex, and almost reached the median surface of the frontal lobe. The abscess cavity contained about three ounces of thick creamy pus. The tympanic cavities and petrous portions of each temporal bone were normal. There was no suppuration in either orbit, but a small quantity of pus was found in the upper ethmoidal sinuses of the nose, on the right side. The left sinuses contained no pus. No changes of importance were detected in other parts of the body.

Case 3.—*Tumour compressing left frontal lobe; convulsions; mania; coma.*—Charles W., age 36, came under my observation when house physician at the Manchester Royal Infirmary in 1884. The patient was brought to the accident room late one evening in December of that year in a comatose state. There

were no unilateral nervous symptoms (such as paresis), no signs of injury to the skull, no peculiar smell of the breath, no indications of disease of the heart or lungs. As no definite history could be obtained, in view of the possibility of some narcotic poisoning, I decided to wash out the stomach. This was done without any improvement following, and the man was afterwards admitted as an in-patient under the care of Dr. Leech. He recovered consciousness gradually next day and was discharged a few days later.

The patient returned to his work and continued fairly well, with the exception of mental dulness, drowsiness and constipation, until June 1, when he had a severe convulsion. He recovered rapidly and after the convulsions ceased, he walked to the Infirmary and was admitted as an in-patient June 1, 1885. From the notes of my friend, Dr. Wild, who was then house physician, I learned that for the first two days after admission he presented no mental symptoms, but on the evening of June 3, symptoms of acute mania developed. He became violent and had delusions of suspicion; later he became more quiet, but remained irritable. On June 4, being allowed to go into the lavatory, he locked the door and jumped through the window. He fell on the grass beneath and immediately got up and commenced to run away. He was seen and captured and brought back to the Infirmary. Beyond a slight bruising of the back, he appeared to have been uninjured. Epileptiform convulsions then commenced and were frequently repeated. In twenty-four hours (from June 5 to 6), he had forty-seven epileptiform convulsions. Between the convulsions he remained comatose. The urine and fæces were passed in the bed involuntarily.

The fits continued for a few days and then gradually ceased, and the patient regained consciousness. He appeared to be in great pain, and frequently made a peculiar wailing noise. Retention of urine came on and a catheter had to be used. In two days after the commencement of the use of the catheter the urine became ammoniacal and contained pus. The patient then improved, the fits ceased, he became conscious and was able to take his food and to pass the catheter himself. The bladder was washed out with a solution of boracic acid daily and the urine became clear and acid. The patient, however, began to emaciate rapidly, and the temperature became raised above normal and irregular. He became much weaker and the retention of urine was succeeded by complete incontinence. He refused food, appeared to suffer from great pain in the head, and often gave a

peculiar wailing cry. The tongue became brown and the teeth covered with sordes. Picking at the bed clothes was noticed, and the patient became completely comatose. Death occurred on July 1.

Autopsy.—Growing from the inner aspect of the dura mater near the anterior portion of the left frontal lobe, was a tumour the size of a large walnut. This tumour had grown into the apex of the left frontal lobe, pushing before it the pia mater which lined the cavity in which the tumour was situated. The brain substance around was not at all infiltrated by the growth, which was circumscribed and could be shelled out easily from the depression in the frontal lobe. On section the tumour was firm and of a reddish grey colour, and contained numerous small spaces (the size of small peas), filled with blood. There was considerable œdema over the surface of the hemisphere, beneath the pia mater. Elsewhere the brain and medulla presented no abnormalities. There was slight tubercular disease at the apex of each lung. Both kidneys were studded with numerous dark red patches, the size of penny pieces. In the liver was a very vascular, dark red, new growth, the size of a large marble. No other changes of importance were found.

The two following cases did not come under my own observation, but are recorded in the clinical reports of the Manchester Royal Infirmary and have never been published.

Case 4.—*Tumour of left frontal lobe, deviation of eye and head to the left; long attacks of profound "sleep."* Ed. B., admitted as an in-patient at the Manchester Royal Infirmary, August 1, 1887, under the care of Dr. Steell.

Previous history.—Five and a-half years previous to admission a boy threw a piece of coal at the patient's head. This struck the occipital region, a scalp wound was produced, but the patient did not lose consciousness. Just when the wound was beginning to heal (five or six weeks after the blow) the patient had a fit. Six months later a second fit occurred and the patient has had five or six fits since. Three months ago he began to be troubled with headache, loss of appetite, sickness and diarrhœa, and his medical attendant ordered him to bed. He slept for two or three days, and it was scarcely possible to rouse him. Similar attacks have occurred once or twice a week since. For several weeks before admission patient is said to have dragged his legs. On July 25 his mental condition became worse, and he was unable to stand. The speech was affected for

about a week before admission. The patient stammered and "made mistakes," "said things the wrong way about." He has complained of pain in the neck for two months.

Present state.—Patient lies with his head and eyes turned to the left. He is very irritable, does not answer questions, appears to be semi-conscious, moans when touched or disturbed. Double optic neuritis is present.

August 4.—Head turned to the left; neck rigid; appears to have much pain in the neck when an attempt is made to turn the head to the right. No paralysis of limbs. Appears to have much pain when the legs are moved; knee-jerks increased.

August 5.—Patient lies with his head still turned to the left. The eyes are half open. When asked "How are you?" he usually answers indistinctly, "I'm all right." There is still much pain when one attempts to rotate the head to the right. He is able to show the teeth and to protrude the tongue quite well. The temperature rose to 104·4° on the evening of August 6; pulse 128, respiration 50. On the morning of August 7 it was 101·2°, in the evening 103·8°. Death occurred on August 8.

Autopsy (*head only*).—Dura mater normal; pia mater congested, but no signs of lymph or pus at any part thereof. The left frontal lobe was occupied by a new growth which extended to the outer and inner surfaces of the lobe. It projected markedly on the median surface and compressed the median surface of the right frontal lobe. Posteriorly it extended to the anterior part of the left caudate nucleus, but the greater part of that nucleus, together with the lenticular nucleus and internal capsule, was not affected. The growth was very soft and vascular, and the greater part had undergone mucoid degeneration. Other parts of the brain were normal to the naked eye.

Case 5.—*Tumour of right frontal lobe; absence of knee-jerks; occipital headache.* Thos. B.[1] aged 17, admitted June 26, 1888, under the care of Dr. Dreschfeld.

Previous history.—Six years before admission patient began to suffer from epileptic fits. For the first two years he suffered from attacks of *petit mal*. These occurred once or twice a week. Four years ago he began to suffer from attacks of *haut mal*. These attacks occurred sometimes two or three times a week; sometimes he would have four or five attacks daily, and then the attacks would cease for a while. Before each attack patient had an aura, consisting of a "twiching sensation,"

[1] Abstract from notes by Mr. Gore.

running up the left arm from the tips of the finger; this sensation would last five minutes before each attack. Three months before admission the fits ceased. Since that time he has been troubled with pain in the occipital region, which sometimes has passed over to the frontal region. The pain, of a throbbing character, has been constantly present. The vision has become affected considerably; for three months he has suffered from intolerance of light, and on looking at any white object there has been frontal headache. Recently he has been troubled with sleeplessness and loss of memory.

Condition after admission.—The patient lies in a drowsy condition, but is easily roused. The face has a languid expression. The pupils are somewhat dilated, and there is an internal squint of the left eye. He is fairly well nourished. He is intelligent; he answers questions somewhat slowly, but correctly. He suffers from giddiness and constant headache in the inferior occipital region. When he looks at anything white he suffers from frontal headache. The pain in the occipital region is of a throbbing character. There is slight paralysis of the lower facial muscles of the left side, and the left orbicularis palpebrarum is feebler than the right. The movements of the left arm and leg are somewhat weaker than those of the right. The tongue is protruded straight. The epigastric, abdominal cremasteric reflexes present on both sides, better marked on the right side than on the left. Plantar reflexes increased. Knee-jerks *absent*. There is no anæsthesia. The gait is staggering; the patient tends to fall towards the right side, and has a sensation of vertigo. He cannot stand upright with his eyes closed. Nothing of importance detected on physical examination of the heart and abdomen. Urine: no albumen, no sugar. Slight dulness at apex of left lung.

June 29.—Marked double optic neuritis.

July 4.—Patient was seized with an attack of rigidity of the muscles of the body, the head was thrown back, the arms, legs, and back were rigid. The attack lasted for about one minute; the patient never lost consciousness completely.

July 13.—Patient has complained of severe headache during the last two days; he complains of pain shooting from the occipital to the frontal region. He is in a drowsy condition most of the day.

July 30.—Patient is very drowsy, almost comatose. He answers questions slowly. Complains of much pain on pressure over the back of the neck. Muscles of the neck rigid; head retracted. Death on August 6.

Autopsy.—Cicatrix of healed phthisis at apex of right lung. A small tubercular cavity at apex of left lung, and several small patches of consolidation around.

Brain.—In the right frontal lobe was a roughly spherical tumour two and a half inches in diameter, which came to the surface in the region of the first and second frontal convolutions; other parts of the brain not affected. Cerebellum normal. No growth at the base of the brain. The growth was *firm*, but was not hard except at the centre. The periphery was of a pale greyish red colour and vascular; the centre was pale and somewhat caseous in appearance; at various parts were masses of calcareous material. The growth could be easily shelled out from the surrounding softened brain substance.

The vertebræ, spinal dura mater, and spinal cord were all carefully examined with negative results. No changes of importance in the other organs.

Remarks.—In two of the cases reported it is interesting to note that optic neuritis was unilateral; in one case on the side of the lesion, in the other it was unilateral though *both* frontal lobes were invaded by tumour growth. Another point of interest is the absence of knee-jerks in three of the five cases above reported. In one of these cases the spinal cord was examined microscopically, and was found to be normal, and it is probable, from the clinical history, that in the other cases the cord would have been found normal had a microscopical examination been made.

The severe pain in the occipital region in Case 5 was somewhat peculiar. In Case 4 the deviation of the head and eyes towards the side of the lesion was a symptom of interest, since Ferrier and others localise the centre for this movement to the præ-frontal region. The very long attacks of "deep sleep" are also of interest in this case.

All these points will be referred to subsequently, since the symptoms of the five cases above recorded are included in the following analysis.

Analysis of the Symptoms, &c., in 50 Cases of Tumour or Abscess involving the Pre-frontal Lobes.

The cases comprise the 5 reported above, and 45 recorded in medical literature in recent years.[1]

Nature.—Of these 50 cases 4 were cases of abscess, and the rest tumours of various kinds—10 of the latter were gliomatous,

[1] For references see list at the end of the paper.

7 sarcomatous, 5 gliosarcomatous, 4 syphilitic, 3 endotheliomatous, 2 tubercular.

Region.—17 involved the right lobe, 22 the left, and 11 both lobes. The convex cortex was the seat of the growth in 18 cases, the median cortex in 1, the inferior cortex in 2, the white matter in 8, the convexity and the white matter in 5. In 3 the growth was at the tip of the anterior part of the frontal lobe; in 3 it was between the lobes; and in 10 it was situated at the base and compressed the frontal lobe.

Symptoms.

Headache was a prominent symptom in most cases. The pain was generally most intense in the frontal region, in some cases in the occipital region. In the records of 50 cases headache is not mentioned, or the locality not stated, in 23; in 1 there was no headache; in 1 the region of the pain varied. In the remaining 25 cases the distribution of the headache was as follows:—

	Cases.
Frontal region only, in	11
Frontal chiefly, occipital also	4
Occipital chiefly, frontal also	2
Occipital at first, frontal later	1
Occipital	2
Frontal and vertical	2
Vertical and temporal (near the growth)	1
Temporal (growth on opposite side)	1
Headache "on the side of the tumour"	1

* In 9 of the 25 cases pain was in the occipital region, with or without frontal headache = 36 per cent. of cases in which headache was *localised*.

Frontal headache is therefore most frequent, but it is remarkable that the occipital headache also should often occur. It is well known that in cerebellar tumour the headache is often occipital, and not infrequently it is frontal or frontal and occipital. Dr. Purves Stewart (*Edinburgh Medical Journal*, January, 1896, p. 659), has called attention to unilateral frontal headache in cerebellar tumour situated in one lateral lobe, the headache in the frontal region being on the side opposite to the cerebellar lesion.

Tenderness on Percussion of the Skull.—In 30 out of the 50 cases there is no note with regard to this symptom. In the other 20 cases it is stated, that there was tenderness on percussion of the skull in the *frontal* region in 15 cases. In one of the cases the occiput was tender also, but the tenderness was most marked in the frontal region. In 5 cases it is stated that there

was no tenderness of the skull on percussion. Of those 15 cases in which there was tenderness on percussion of the skull, the tumour growth was situated at or near the region of the tender area in 12. In 1 there was tenderness on one side, and both frontal lobes were the seat of tumour growth.

Hence it appears that if tenderness be present on percussion, it is of value as an indication of the region of the skull in which the growth is situated. Also when tenderness is present in the frontal region, if it be unilateral, the growth is generally on that *side*.

Tenderness on percussion is not a sure indication that the growth is cortical, though this is generally the case. Thus in the 15 cases in which there was tenderness on percussion the growth was superficial (cortical or in the meninges) in 12, but in 3 it was in the white matter. Again, in the 5 cases in which there was no tenderness the growth was cortical in 3, seated in the white matter in 2.

Ophthalmoscopic Examination.—In the 50 cases analysed, the records of ophthalmoscopic examination were as follows:—

	Cases.
Double optic neuritis, in	25
One eye previously lost; optic neuritis in the remaining eye, in	1
Vision impaired in both eyes, in	1
Vision lost in both eyes, in	1
Condition of optic discs and vision not mentioned, in	11
Optic discs normal, in	4
Optic neuritis on one side only (the side of the lesion), in ..	1
Optic neuritis unilateral, lesion in *both* frontal lobes, in	1
Optic neuritis very much greater on side of the lesion, in ..	2
* Blindness on the side of the lesion; the other eye only affected at a later date, in	1
Well marked primary optic atrophy on the side of the lesion; slight optic neuritis on the other side, in	2

* Hence in 7 out of the 35 cases in which ocular changes were noted, the failure of sight or the ophthalmoscopic changes were on one side only, or were very much more marked on one side. In 6 of these 7 cases, the marked ocular changes were on *the side* of the lesion; in one case there was unilateral optic neuritis and a bilateral lesion.

This marked unilateral tendency, or marked difference on the two sides, in 7 out of 35 cases is of some interest, since such a difference does not occur, or does not occur in this proportion of cases, in lesions of other parts of the brain substance.

Sense of Smell.—In 33 out of the 50 cases no note with respect to the sense of smell is given. In 10 cases it is stated that smell was not affected. In 7 there was unilateral or bilateral loss of smell, and in these cases the growth involved the basal region, *i.e.*, region of the olfactory nerves.

Loss of smell, if present, is therefore an important symptom, but if the sense of smell be normal no conclusion can be drawn from this fact alone.

Exophthalmos.—This symptom was bilateral in 1 case, unilateral (on the sides of the growth) in 2 other cases. In all these cases the growth had invaded the dura mater and anterior fossa of the skull.

Localised Swelling in the frontal region of the skull was recorded in one case; in another case there was an external swelling in the temporal region, at the lower angle of the frontal lobe.

Motor Symptoms.—Paresis, or paralysis of the face or arm or leg, or of all three parts, on the side opposite to the lesion, was met with in many cases at some period; but in the majority it was very slight, and was often noticed *only* at the end of the illness. The paresis was due to the extension of the growth backwards from the præ-frontal towards the motor area of the cortex, or to softening around the tumour growth implicating the motor area or the motor fibres coming from that region.

	Cases.
Slight paresis on side opposite to the lesion, noted at some period, in	27
Marked paralysis, in	4
Motor symptoms absent, in	5
Motor symptoms not mentioned, in	14

Convulsions were met with in many cases; their nature is shown in the following table. The frequency of the attacks varied greatly.

	Cases.
General convulsions occurred at some time in the course of the patient's illness, in	14
Unilateral convulsions from involvment of the motor area of the cortex, in	10
*Convulsive attacks (clonic spasms) in both arms, in	3
Fits resembling those of hystero-epilepsy, in	1
Fits beginning by the legs being drawn up into the lithotomy position, in	1
Attacks of tonic spasms in the neck	2
Convulsions absent, in..	4
Convulsions not mentioned, in	15
	50

*In two of these cases the growth was median and involved the median surface of both frontal lobes. In one case there was a lesion in the præ-frontal area, near the arm centre, in *each* hemisphere.

Anæsthesia was never recorded; in many cases it is definitely stated that sensation was not affected, in others no note is made.

Ataxia.—Bruns [20] has drawn attention to the fact that ataxia resembling that of cerebellar disease is sometimes met with in lesions of the præ-frontal area.

In the 50 cases analysed, ataxia, unsteadiness in walking, or a reeling gait was present in 14; these symptoms were not mentioned in 33, and in 3 cases it was stated that no ataxia was present.

Knee-jerks.—It has been long known that the knee-jerks are sometimes absent in cerebellar lesions, whilst in other cases they are increased: why this difference should occur is not known. It is interesting to note that the knee-jerks are sometimes absent in frontal tumour also; in the 50 cases the condition was as follows:—

	Cases.
Knee-jerks absent on both sides, in	6
Knee-jerks very feeble on both sides, in	2
Knee-jerk very feeble on side opposite to tumour, in	4
Knee-jerk very feeble on side of tumour, in	2
Knee-jerks normal or increased, in	16
Knee-jerks not mentioned, in	20

In 30 cases only was the condition of the knee-jerks referred to, and the proportion of cases in which they were absent, amongst these 30 cases was 20 per cent. (6 in 30).

In all the cases in which the knee-jerks were absent, there was no indication of the lesion of the spinal cord or peripheral nerves, but in only one of the cases, the first reported in this paper, was the absence of microscopical changes verified by pathological examination. The absence of knee-jerks is a symptom of interest. In tumour growths involving the motor region of the brain, the knee-jerk on the opposite side is generally increased, and on the side of the lesion, present, if not increased. In 30 of such cases taken for comparison with the 30 lesions of the præ-frontal area analysed above, I found the knee-jerks both present in all except one, and in this case the growth was very extensive and invaded the basal ganglia. The connection of each lateral cerebellar lobe with the præ-frontal region of the opposite side,

is of interest with respect to the loss of long knee-jerks that sometimes occurs in lesion of both these regions.

Mental Symptoms.—The psychical symptoms which have been observed after removal of the frontal lobes in animals are referred to at the commencement of this paper, and it is interesting to note, that clinically mental symptoms have been noted in most cases of lesions of the frontal lobes, though the exact nature of these symptoms has varied somewhat. A dull mental condition, mental decadence, loss of attention, a drowsy, and semi-comatose state have often been recorded.

Some writers have attached considerable importance to a childish behaviour of the patient, with an abnormal tendency to fall asleep.

Jastrowitz[23] has described a peculiar mental condition which he has met with in cases of lesion of the præ-frontal lobes, a condition of mental impairment or dementia along with a peculiar cheerfulness or hilarity, and tendency to jest and make puns (*Verhandlungen des Vereins f. innere Medicin*, Berlin, 1888). He acknowledges that this mental condition is not met with in all cases, and that it occurs also in other diseases, such as general paralysis of the insane and in chronic alcoholism; but he thinks that in a case presenting general symptoms of intra-cranial growth, the tumour may be localised in the frontal lobe, if this peculiar mental condition should be present.

Oppenheim[23] also draws attention to this peculiar humorous mental condition in cases of frontal tumour, and points out that the cheerful mental state stands out in sharp contrast to the miserable physical condition of the patient. There is a tendency to joke, to make sarcastic or trivial answers; Oppenheim compares the mental condition to that of a patient awaking imperfectly from a pleasant narcosis. He adds that it must be allowed that this mental condition occurs principally in tumours of the frontal lobes.

Bruns[20] also describes a similar mental state.

Welt (*Deutsches Archiv. f. Klin. Med.*, Bd. 42, 1888, p. 339) attaches importance to an irritable mental condition, with violent and mischievous behaviour, especially in lesions of the convolutions on the orbital surface.

Lloyd (*Journal of Nervous and Mental Diseases*, p. 81) draws attention to a peculiar "inhibition of thought; an increase of the time reaction of cerebration;" a peculiar slowness in answering questions, though the answers are quite intelligent.

In the fifty cases which I have analysed, mental symptoms were generally well marked; in many cases they were the earliest and most prominent symptoms. The following table shows the varieties of mental condition met with :—

	Cases.
* A condition of mental decadence; a dull mental state; loss of power of attention; loss of memory; loss of spontaneity; the patient taking no notice of his surroundings; sleeping during the greater portion of the day, or being semi-comatose	32
Loss memory, mental failure, but patient cheerful	6
Patient suspicious; suffered from delusion, and was occasionally violent	1
Patient irritable and violent	1
Patient generally asleep; irritable when awake	2
Patient ambitious, excitable, memory lost	1
Slowness of mental processes; patient simple and childish ..	1
Mental anxiety; childishness; hallucinations; suicidal tendencies	1
Mental condition not stated	5
	50

* In two of these it is noted that the patients were in a perplexed mental condition, and constantly appeared to be searching for something.

In some cases in which the mental symptoms were well marked, the lesion was chiefly in the white matter, in some cases, chiefly in the cortex, whilst in others both cortex and white matter were involved; so that one does not feel justified in drawing any conclusion as to whether the mental symptoms are the result of lesion of the cortex or of the white matter. In 3 cases in which the lesion was chiefly cortical, the mental symptoms were comparatively slight.

Loss of *power* in the *muscles of the back* is not mentioned in any of the records.

Sudden onset of Symptoms.—Sudden coma, when only slight and trivial symptoms had been complained of previously, was recorded in 3 cases.

Attacks of Coma.—As a rule the patient finally became

comatose, and in many cases was comatose or semi-comatose for a long period before death. But apart from this terminal comatose state, in some cases the patient suffered from one or more previous attacks of coma, without any preceding convulsions. Sometimes these attacks were followed by hemi-paresis; in other cases no paresis followed. Attacks of unconsciousness previous to the final coma were noted in 10 out of the 50 cases analysed.

Remarks on Diagnosis.—In a case in which there are general symptoms of intra-cranial growth or abscess, the localisation to the præ-frontal region often presents considerable difficulty. The diagnosis is sometimes especially difficult between cerebellar and frontal tumours. In tumours of the præ-frontal region, whilst the headache is chiefly frontal, very often there is occipital headache also. In cerebellar tumours whilst the headache is chiefly occipital, still not infrequently there is frontal headache. In tumours of the cerebellum the knee-jerks are sometimes absent, but as shown by the cases recorded in this paper, the knee-jerks are sometimes absent in lesions of the præ-frontal region. Also ataxia, simulating the ataxia of cerebellar disease, may occur in lesions of the præ-frontal region. Each præ-frontal lobe of the cerebrum is connected with the lateral lobe of the cerebellum of the opposite side, by fibres passing downwards in the anterior part of the internal capsule, on the inner side of the crus cerebri, and in the superior cerebellar peduncle; and this connection may, in some way, account for the above-mentioned symptoms being often common to lesion of both the præ-frontal region and the cerebellum.

In many cases, however, a careful study of the symptoms will lead to a correct localisation. Mental symptoms (especially mental dulness and degeneration) are prominent, and often early symptoms in lesions of the præ-frontal regions, whilst they are generally slight and occur late in lesions of the cerebellum. Paresis on one side—hemi-paresis, or paresis of leg, arm or face only—occurs in a large number of cases of præ-frontal lesions at a late period, owing to the extension backward of the growth, or of the surrounding

softening; whilst paresis of the arms or legs is very rare in cerebellar lesion. Convulsions, general or localised, often occur at some period in præ-frontal lesion; very rarely in cerebellar lesion.

Tenderness on percussion or pressure over the frontal region appears to be a sign of value in localising the lesion to the præ-frontal region.

Whilst optic neuritis is often bilateral in præ-frontal tumour and abscesses, still the facts above recorded show that in some cases it is unilateral, or it is *very much* more advanced on one side, or vision is *very much* more impaired on one side, or there is primary optic atrophy on one side, and only slight optic neuritis on the other. I have never met with any case of cerebellar tumour in which there was this tendency to very marked affection of one optic disc, whilst the other was normal, or only very slightly affected.

[It is interesting to note that the lesion has been on the side on which the optic disc was markedly affected in 6 out of 7 of these cases; in the seventh the lesion was bilateral]. When present, this marked difference of the two optic discs appears to be of some diagnostic importance.

Localised swelling in the frontal region, unilateral exophthalmus and loss of smell are rare symptoms, but, if present, form important evidence in favour of a lesion of the præ-frontal region.

It is to be noted, however, that loss of smell has been reported in cerebellar tumour, but of course is exceedingly rare, whilst it is reported in 7 out of 17 cases of præ-frontal tumours.

With regard to the diagnosis between lesions of the præ-frontal region and the so-called motor area, evidence in favour of localisation in the former would be the prominence and early onset of mental symptoms; the absence or slight nature of motor symptoms, and the development of these symptoms, when present, at a late period, after the general and mental symptoms have become well-marked. Whereas in lesions of the motor area, mental symptoms are often absent or slight until a late period; paresis is an early symptom and develops into well-marked paralysis. Also the

tenderness or percussion over the frontal region, and (in a few cases) the absence of knee-jerks, the loss of smell, the unilateral nature of the ophthalmoscopic changes, or very marked difference of these changes on the two sides, would be points in favour of a lesion of the præ-frontal region.

Suitability for Operative Treatment.—In the 50 cases analysed, the 4 cases of abscess were all suitable cases for surgical interference. One case,[32] McEwen's, was operated on with success. Seven cases of tumour of the præ-frontal lobes were so suitable and of such a moderate size, *even at the time of the autopsy*, that they could have been easily removed by surgical operation. In 2 other cases, a tumour was removed; in one case successfully (Durante), in the other unsuccessfully.

References to the Forty-five Cases included in the above Analysis of Fifty Cases.

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(2) Reinhard. *Inaug. Dissert.* Berlin, 1893. "Zur Pathologie der Stirnlappentumoren."

(3) Obici. *Il policlinico.* Epitome, *British Medical Journal*, August 10, 1895.

(4) Galavielle and Villard, *Arch. de Neurologie*, July, 1895.

(5) Raymond. *Arch. de Neurologie*, Oct., 1893.

(6) Ferrier. *Lancet*, June 4, 1892.

(7) Bury. *Medical Chronicle*, April, 1893.

(8, 9) Bouveret. *Lyon Médical*, Oct. 27, 1895 (two cases).

(10) Bramwell. *Edinburgh Medical Journal*, Jan., 1887.

(11) Durante. *Lancet*, Oct. 1, 1887.

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(17) Starr. (Allen). *American Journal of Medical Science*, April, 1893.

(18) Bramwell. *Edinburgh Medical Journal*, Jan., 1879.

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(20, 21, 22) Bruns. *Deutsche medicinishe Wochenschrift*, No. 7, 1892 (the second, third, and fourth cases).

(23—31) Oppenheim. *Archiv. für Psychiatrie*, vol. xxi. (nine cases).

(32) McEwen. "Pyogenic Infective Diseases of the Brain and Spinal Cord," Glasgow, 1893.

(33) Bouveret. *Lyon Médical*, Dec. 8, 1895.

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(35) Booth. *Journal of Nervous and Mental Diseases*, 1893, p. 70.
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(42) McDowall. *Journal of Mental Science*, vol. xxx., p. 87.
(43) Obernier. "Ziemssen's Encyclopædia," vol. ii., p. 264.
(44) Knapp. *Archives of Ophthalmology and Otology*, vol. iv., p. 245.
(45) Phillipson. *Medical Times and Gazette*, 1882, vol. ii.

A CONTRIBUTION TO THE PATHOLOGY OF EPILEPSY. A REPORT OF TWO CASES IN WHICH A PORTION OF THE BRAIN CORTEX WAS EXCISED AND EXAMINED.[1]

The Microscopical Examination of the first Case by Dr. A. Wiener.

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A CAREFUL study of a large part of all that has been written on the value of trephining for epilepsy has convinced me that it is quite impossible to gather from the literature alone what the value of operative interference in epilepsy may be. This is so for many reasons. In the first place, the significance given to the designation "operation for epilepsy" is a variable one. Leaving aside the operations that are done on other parts of the body such as the eyes, the nose, &c., for the cure of epilepsy by removing so-called reflex sources of the disease and confining our criticisms to operations on the skull, we note that for one writer operation in epilepsy means simple trephining; for another, trephining and removal of the dura; while for a third, it means not only both of these procedures, but in addition, the excision of a portion of the cortex, whether or not it be the seat of apparent disease. When such radically different interpretations are put upon the words "operation for epilepsy," it will be seen how extremely difficult it is to gather any reliable data as to the value of what we consider operative procedure in epilepsy; that is the value of making a large opening in the skull, of dissecting the dura, and of removing

[1] Read before the American Neurological Association, June, 1896.

the cortical area which has been shown by clinical observation and experimental test to be the seat of the lesion.

At the present day there would seem to be a tendency to absolutely decry the value of all forms of operative procedure in the treatment of epilepsy. This attitude of the profession is the natural reaction to the intemperate espousal of the supposed usefulness of operation which occurred a few years ago. Unfortunately, during this over-enthusiastic period in the early development of brain surgery, many cases of epilepsy were operated on, in one of the three ways mentioned above, in which there was no indication for operation, and in which no hypothesis could be formulated to explain why such an operation could be of benefit. These cases have contributed to establish the verdict of "no utility."

Arrays of statistics have been gathered by different writers to prove or to disprove the value of operation on the skull in epilepsy; but in almost every instance there is such utter lack of discrimination in selecting the cases that should be included, or excluded, that such tabulations are worse than useless; they are positively baneful, for by manipulation they may be made to bear evidence for or against the value of operation. In addition to this charge, the hackneyed one of premature report of cases that have been operated on must be mentioned. It is so ridiculous for a man with scientific training to report the favourable or unfavourable issue of a case in which operation on the head for the relief of a disease like epilepsy has been done, a few weeks after the operation, that one is chagrined even to have to refer to these cases. Yet a large number of such cases have been, and are reported, and statisticians to whom the critical faculty is as foreign as is the nature of evidence, use such cases to settle the question of value of trephining operations!

I would not be understood as saying that there are no cases on record in which indications for operation in the sense in which we use the term, viz., making a large epicranial bone flap, dissecting off the dura and excising a piece of the cortex, have not been clearly defined, and in which the operation has been masterfully done. There are

such cases, but they comprise the great minority in the literature on the subject.

The writer believes that the indications for operation in epilepsy are very limited, but that such indications occur not only in Jacksonian epilepsy, but in so-called idiopathic epilepsy.[1] Operation is indicated in Jacksonian epilepsy when there are evidences of irritation of a circumscribed motor cortical area, and it matters not what such irritation may be caused by. The source of irritation, as well as the surrounding cortex for a considerable distance, should be extirpated, and the sooner such extirpation follows the first attack of localised spasm, the greater will be the chances of absolute cure.

Operation is indicated in idiopathic focal epilepsy when the convulsive attack begins always with the same localised spasm, and especially if, in the early stages of the disease, such localised spasm be the only convulsive manifestation.

It is the purpose of this paper to elicit your criticism of two cases, the first conforming to the type of Jacksonian epilepsy, and the second to that of idiopathic epilepsy.

The points to which I wish particularly to direct attention in the first case are: the careful clinical study, the exact location of the lesion, the early operation—for the patient had no more than three attacks altogether, and only one of them passed into a typical epileptic convulsion—and, lastly, to the pathological changes which were found in the brain, and the meninges and the causal relationship existing between them and the attacks of epilepsy. The transference of the recently removed cortical matter directly into absolute alcohol, thus avoiding to as great extent as possible the formation of artificial changes, and the use of the most approved technic for the detection of changes in the cortex, were important factors in contributing a satisfactory explanation of the case. The probability of complete cessation of

[1] The use of the term, idiopathic epilepsy, for these cases is deprecated, for idiopathic epilepsy is used synonymously with inherited epilepsy by most writers. If these cases could be called focal epilepsy, insomuch as the motor or sensory discharge of energy points to a localised area in the brain, without confounding them with Jacksonian epilepsy, which is always focal, and in which some lesion, apart from the supposed change in the large cells of the pyramids, exists, it would be very desirable.

the symptoms of epilepsy, now that the diseased area is removed, will also be touched on.

Case I.

History.—O. L., 20 years old, an American of German parentage, consulted me April 19, 1895. The family history showed on the maternal side mental trouble. The patient had one younger brother defective mentally, who died of spinal disease. The patient is the fourth child of the family. During infancy he was free from convulsions and serious disease. He went to school until his fifteenth year, and was considered moderately bright. He had scarlet fever and diphtheria six years ago and made a good recovery from each of them. He was well till November 29, 1894. At that time he was working in a theatre selling librettos. On the evening of that day while sitting home reading a newspaper, he felt a sensation arising in the fingers of the right hand like an electric shock, associated with a twitching of the thumb and index finger. This lasted about three minutes. The sensation went up to the side of his face, then he lost consciousness. He had in short, a typical epileptiform attack. There was no frothing at the mouth, but he bit the tongue. After the attack he was prostrated and wanted to sleep. He felt so bad that he did not go to the theatre that night. His attack was looked upon as nervousness and fright but, nevertheless, his parents kept him home for several days. Two or three times that week he had some twitchings of the fingers, less severe than the first and not accompanied by loss of consciousness. He had no further trouble or complaint until two or three weeks before coming to me, that is in March 1895, when he experienced a numb feeling in the right hand, this would come and go quickly. He says the sensation was like a shock; all the strength, the natural feeling seemed to go out of the hand. Three days ago, while walking in the street he felt twitchings in the right hand, accompanied by the same numb feeling which he had before experienced. The twitching lasted ten minutes and was confined principally to the index finger and thumb. A premonitory pricking sensation of a few seconds

duration seemed to herald it. Then after a short time a second attack of the twitching occurred. He did not lose consciousness but felt bad after the attack.

There is no history of recent injury to head of any import. Some four or five years ago while practising in a gymnasium he was thrown against the wall, and although stunned for a few minutes there was no loss of consciousness nor was there headache following it.

Examination.—No defect of station or gait. Both knee jerks lively, right exaggerated. Considerable ankle clonus on right side, absent on left. Grip of right hand weaker than left and weaker than formerly. Triceps reflex on right side livelier than on left. Myotatic irritability of entire right half of body greater than left. Does not complain of cephalic parnesthesiæ or headache. There are no scars on the surface of the head. No tenderness on percussion of scalp. Has some marks of degeneracy such as vaulted palatine arch, non-lobulated ears, clubbed finger-ends, and coarse hair. There is slight tremor of the right hand and a little uncertainty of movement, not sufficient, however, to be called ataxia. Sensibility of right hand and right upper extremity, in fact, of every part of the body seems to be intact. The patient was referred to Dr. Knapp, for examination of the eyes, who reported as follows :—

The patient, Mr. L. whom you sent to me for examination, shows the following conditions :—

V. $\frac{20}{40}$ brought up to $\frac{20}{20}$ by glass correcting a low degree of myopia and myopic astigmatism (R. 0·50 sph., L. 1·00, cy. 1·80 ; muscles out of balance, patient showing a small amount of esophoria both for distance and near.

Muscular and refractive condition unaffected by complete relaxation of the accommodation with homatropine. Field for white and for all colours normal in each eye. Fundus normal in ophthalmoscopic examination both with and without a mydriatic.

A diagnosis of Jacksonian epilepsy was made, and on account of the absence of the cardinal symptoms of brain tumour, such as optic neuritis, headache, vomiting, etc., the pathological diagnosis was meningo-encephalitis. Operation

was urgently advised. As it did not seem to the parents that the boy was ill there was considerable hesitancy and delay on their part in consenting to such a procedure.

In the meantime the patient was taken to Dr. B. Sachs, who made the same diagnosis and recommended the same measure for relief. The patient was admitted to Mount Sinai Hospital on May 9, Dr. Gerster having consented to perform the operation. The patient was prepared in the usual manner, and the topography of the right hand area particularly the thumb and finger area was outlined on the left side of the skull. Dr. Gerster proposed to do the bone flap operation. The skull was found to be extremely thick, and the procedure of cutting through it with the mallet and chisel was a very slow one. The patient did not seem to tolerate the ether very well, and frequently it was necessary toward the end of the operation to cease work as it was noticed that after every few blows of the mallet the pulse beat would diminish, in some instances rather alarmingly. It was found impracticable to make a bone flap so the section of bone was removed. The dura was not adherent, and it with its vessels, presented a very normal appearance. The cortex was irritated by a weak faradic current and flexion movements of the hand, exquisitely in the index finger and thumb were produced. On account of the time consumed in getting into the brain cavity and the condition of the patient, it was thought wise to postpone further investigation and operation for a few days. The wound was dressed with a simple aseptic dressing. The patient rallied well, aided by moderate stimulation, and the next day he had an evening temperature of 101·6. On the second day after this preliminary operation he had an attack beginning with twitching in the face and right hand and forearm; this latter lasted for about ten minutes, when he became subject to a rather violent general epileptic seizure, the convulsive part of which lasted about ten minutes. He did not, however, regain consciousness, and after a quarter-of-an-hour he had a second severe convulsive seizure, after which he slowly recovered consciousness. After the preliminary operation the right hand seemed to be quite powerless, although no

part of the cortex had been removed. Sensibility of the right hand remained as before. Four days after the first operation, May 17, the second operation was done. On removal of the bandages it was seen that there was marked œdema of the pericranial tissues extending over the entire right surface of the skull. The wound itself was healthy in appearance. A flap was raised from the dura over an area corresponding to the original opening and it seemed to be quite normal. That part of the central convolutions which on being irritated with the faradic current caused a contraction of the fingers of the right hand was then excised. Two pieces of cortical tissue each about the size of the terminal phalanx of the thumb were removed.

The patient tolerated the second operation very much better than the first, and at neither was any considerable amount of blood lost. The wound was closed by simply apposing the epicranial flaps and covered with the customary surgical dressings. On the day following the second operation the patient felt quite well. Examination showed well-marked paralysis of the right hand, upper extremity and paresis of the right side of the face. Pain sensations could be quite well located in the right hand, contact sensations to a much lesser degree. On the fourth day after the operation he had a mild attack beginning with twitching movements around the right eye and right side of the face, and then becoming general. Later in the day he had a severe general attack in which he was unconscious for about an hour. After recovery from this there was an attack lasting about three minutes, in which the twitching was limited to the right side of the face, nose and eyelid. After this attack the motor paralytic symptoms began to mitigate and within a week they had become comparatively slight. Coincident with the disappearance of the motor paralysis, a paralysis of sensation seemed to develop in the right index finger and thumb, and a very considerable ataxia in the entire hand.

From a surgical standpoint the patient made excellent progress, and was discharged from the hospital June 12th. At no time after the second operation did the temperature

go above 100° F. except on the ninth and tenth day, when the thermometer registered a fraction above that figure.

Examination, July 9, 1895.—The patient is active and bright mentally, and strong physically. He does not make any complaint except of a feeling of weight in the right hand. Gait and station are normal. Dynamometer, right hand 70. Left hand 90. The knee jerks are both lively, the right slow in action but it moves through a large arc. There is no ankle clonus, the right hand and fingers show some ataxia and there is a slight perversion of tactile sense, loss of dexterity in grasping anything such as a knife or in picking up a number of coins in succession, but slight diminution of the sense of position Muscular sense is considerably diminished in the right upper extremity.

July 21. After full warm bath had a *petit mal* attack. Right hand became numb, and the hand drew together with flexed fingers. Speech became thick and inarticulate. One half of tongue, the right, felt numb, and was the seat of a severe pricking sensation. The attack lasted two or three minutes, but there was no dizziness or loss of consciousness, nor did he sleep or become stuporous afterward. Three or four days later he went to see Dr. Gerster who found a strip of gauze in the wound which had prevented it from completely uniting. Since the last visit he has gained four pounds in weight, memory is good, and he is apparently in good shape intellectually.

Dynamometer, right hand 65, left 85. Dexterity, grace, and manipulatory skill in right hand much less than in the left. Has noticed that since the last attack he cannot compress the middle finger of right hand; all the other fingers can be forcibly flexed so that he has a good grip except the middle finger. Tactile sense is lost in this hand. Sense of contact seems to be preserved, that is when a coin is dropped in the right hand he knows when it touches the hand, but he cannot tell when it is removed. Sense of position and muscular sense are likewise injured only in a slight degree. Ataxia of fingers in right hand marked, for instance when told to bring the end of each finger successively in contact with the end of thumb he gets all mixed. Likewise when

he essays to touch the tip of nose. Can feel cotton all over hands. He is taking now thirty to forty drops of a saturated solution of potassium bromide three times a day.

September 2, 1895.—The right hand is becoming less adroit, especially the fingers. The ataxia and certainty of purposive movements of fingers is somewhat greater. He is now taking sixty drops of a saturated solution of bromide at bedtime. This quantity is reduced to fifty. There have been no attacks. He has no sensations of vertigo, mental confusion, or fainting spells. He feels strong and well, and is at work. Memory and other intellectual faculties seem normal. After the operation he noticed that the toe of the right shoe was worn more than left; this is no longer so. He can use the right hand moderately well. Has power in the whole hand, he says, but in the individual fingers it fails him. Dynamometer, right 20, left 90. There is loss of sense position in the right hand and fingers; but this is not complete. When all the fingers are flexed he cannot extend the fingers individually, nor in pairs, they must all be extended together. Extensor force is less than flexor in fingers, and in wrist. Articular sense in right hand is fairly good. The fingers of the right hand have a tendency to remain flexed with closed fist. When he squeezes anything with left hand, the fingers of right flex into palm (showing motorial impulse from left side goes down right). Tactile sense all right; can tell the moment anything touches his hand. Has good sense of weight and temperature. Knee jerks right side slightly exaggerated. Tongue deviates slightly to left. Face not so completely and thoroughly innervated on right side as on left. Triceps reflex right side exagerated, and the myotatic irritability of the right upper extremity is increased. The strength of the lower extremities seems to be equal on both sides. He has been taking twenty-five grains of bromide daily, one half the quantity in the early evening, the other half on retiring.

Examination on June 1 of the present year, somewhat more than a year after the operation, and very nearly a year after the date of the last epileptic attack, shows that the

strength of the right hand causes the dynamometer to register 30, and with the left hand 100. Myotatic irritability of the right upper extremity is increased. There is slight exaggeration of the right knee-jerk, but there is no ankle or patellar clonus. There is no ataxia of gait; the patient stands well with the feet together, and on either foot alone. Examination of sensibility shows that the sense of touch in the right upper extremity is exquisitely preserved, likewise the sense of pain and temperature. Muscular sense in the hand is somewhat diminished, and the sense of position is not perfect; but aside from these there are no sensory shortcomings. The right hand has a tendency to remain in a semi-flexed position, and the fingers cannot be extended individually, but they can collectively. There is very little ataxia of the right hand.

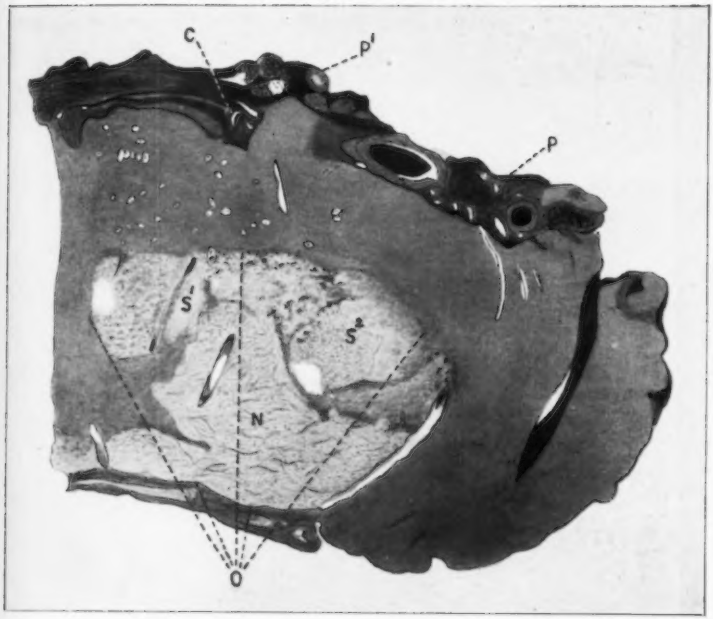
The patient is engaged in clerical work; he has learned to write with the left hand, and, aside from the inconvenience attending from a loss of dexterity with the right hand, he may be said to be in perfect health. During the past four months the patient has been taking twenty grains of bromide of potassium once a day, on retiring; this, with the ordinary measures for the maintenance of a commendable nutrition is all that is being done for him. There have been no attacks of vertigo, no recurring sensations of numbness and loss of strength in the hand, no attacks of mental confusion, nor, in short, anything that could be construed as a motor, or psycho-motor discharge of energy resembling an epileptiform attack. There has been no mental deterioration whatever; in fact, it is probable that the mental faculties of the patient are more active now than they were before the operation.

It may be said, and if so, no effort will be made to controvert it, that the quantity of bromide of potassium which the patient has taken has been sufficient to hold the epileptic attacks in abeyance. If the patient remains in my care it is proposed to keep up the quantity of bromide which he is now taking for at least two years longer, that is, providing that within that time no epileptiform manifestation shows itself. If it should, the dose of bromide will be materially increased. In the opinion of the writer, it would be most

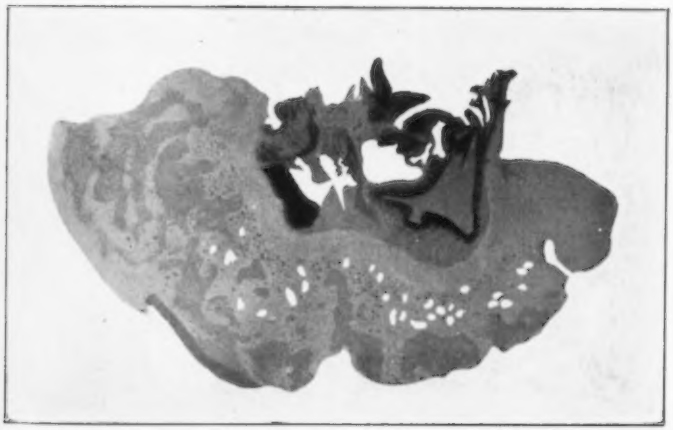
unjustifiable not to continue the use of bromide for a long period after the operation in a case of this kind, even when it is believed that the focal origin of the disease has been entirely removed, as it probably was in this instance. It is thought that by such medicinal measures the motor cells in the neighbouring cortex, which were participants in the few convulsive seizures from which the patient suffered, can be so steadied as to prevent further tendencies to explosion which they would naturally have on account of such explosion having previously taken place.

The cortical mass removed in this case measured about four ccm. in length and one and one half ccm. in thickness. The mass was divided into pieces measuring one half ccm. in thickness, and then placed in absolute alcohol. The Nissl stain was used for study of the cells, while for the tissue proper the hæmatoxylineosin and the borax carmin methods were used.

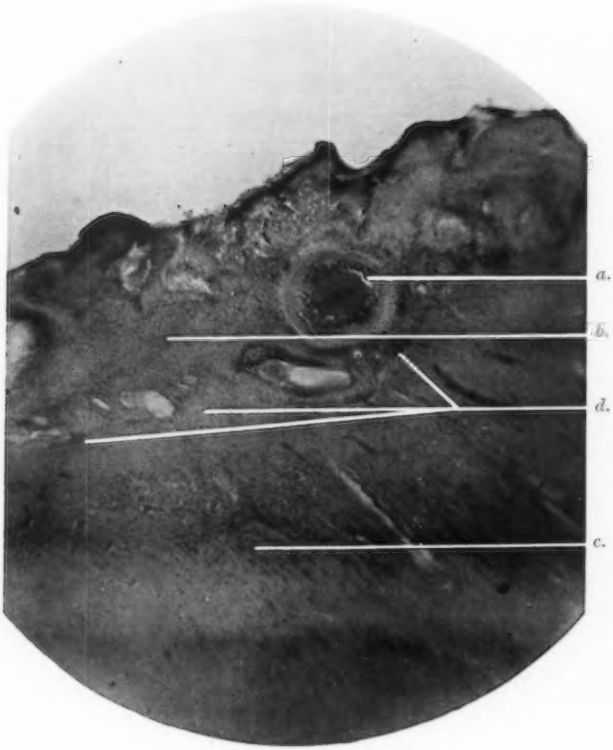
A general view of the specimen taken from the most striking part of the lesion, shows marked pathological change in the pia, in the blood vessels, in the subpial space, and in the cortex, (see fig. 1). The pia is thickened and intimately attached to the cortex; immediately under the pia there appears a diseased cortical area; directly beneath this diseased cortical area there is a fairly normal portion, and then just at the junction of the grey and white matter, there is a distinct area of softening combined with increased neuroglia measuring about one half ccm. in length, and one quarter ccm. in breadth. The pia on closer microscopical examination is seen to be very much thickened; there is considerable formation of connective tissue. The distended blood vessels show thickened walls infiltrated with spheroidal cells. Some of the blood vessels are entirely occluded. Throughout the whole region the pia is closely attached to the cortex; in some places there is an actual growth of connective tissue from the pia into the cortex, completely disorganising it; in other places the pia appears to be merely glued to the surface of the cortex by a large number of spheroidal cells, which shows that in this region a more or less acute process had occurred. This might be accounted



CASE 1.—FIG. 1.



CASE 2.—FIG. 1.
Stained with Picrocarmine showing Hæmorrhages.



CASE I.—FIG. No. 2 × 115.

Photomicrograph, representing area *(p)* of Fig. No. 1. Thickened pia closely attached to the cortex.

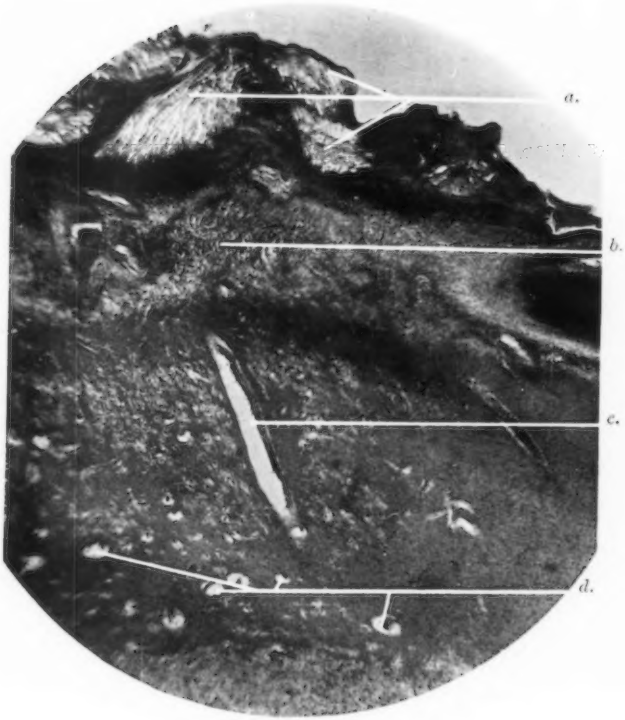
a.—Blood-vessel with thickened wall, and filled with blood.

b.—Pia thickened and infiltrated.

c.—Cortex.

d.—Margin between cortex and pia.

Collotype by Danielsson & Co.



CASE I.—FIG. No. 3 × 115.

Photomicrograph, representing area *(p)* of Fig. No. 1. Pia with hyaline degeneration in capillary blood-vessels.

a.—Capillary vessels which have undergone hyaline degeneration.

b.—Disorganised cortex.

c.—Shrunken blood-vessel with large peri-vascular space.

d.—Same.

Collotype by Davidsson & Co.

CASE I.—FIG. No. 4 × 325.

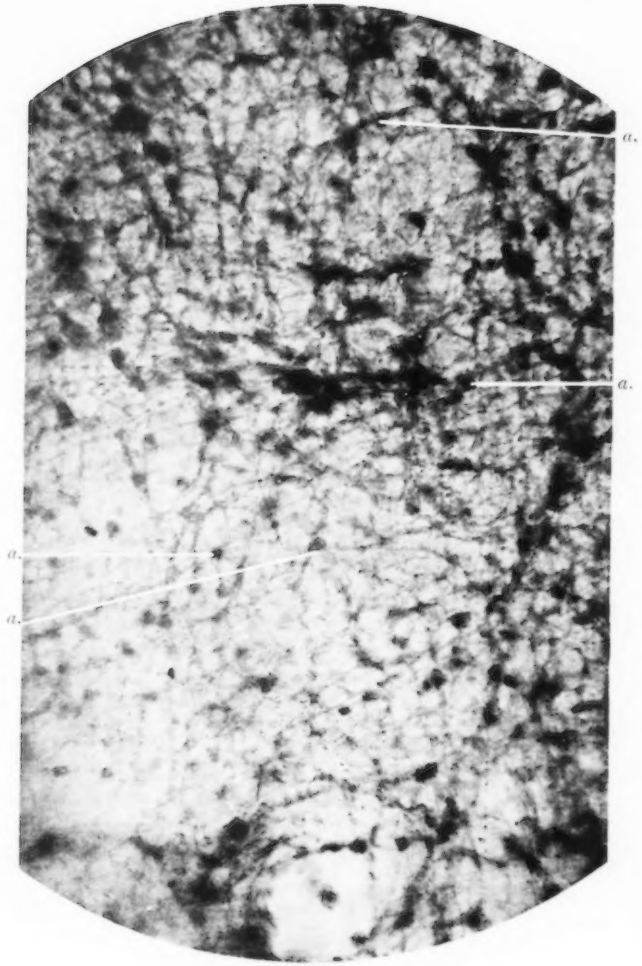
Photomicrograph, representing area (*c*) of Fig. No. 1. Disorganised cortex with pia closely attached.

a.—Connective tissue growth in pia.

b.—Cell infiltration between cortex and pia.

c.—Disorganised cortex.

d.—Dilated blood-vessel filled with blood.



CASE I.—FIG. No. 5 × 325.

Photomicrograph, representing area *(n)* of Fig. No. 1. Neuroglia tissue as seen in the neighbourhood of the area of softening.

a.—Neuroglia cells.

Collotype by Danielsson & Co.

for by supposing that it occurred in the interval between the first and second operations. At one particular point, just opposite to where the connective tissue septa are seen extending into the cortex, there is a small group of capillary blood vessels which have undergone distinct hyaline degeneration (see fig. 3); in this particular portion of the pia we find the densest connective tissue formation (figs. 2 & 3).

Cortex.—The subpial zone is very much disorganised by the infiltration of spheroidal cells (fig. 4), and by septa of connective tissue which have grown down from the pia into the cortex. The blood vessels appear shrunken and some are entirely occluded, and the walls of the vessels are thickened and infiltrated with cells. The perivascular spaces are wider than normal. It is especially in the environs of the blood vessels that the acute process seems to have been most active. Just beneath this subpial area, is a zone of cortical tissue which on casual inspection appears entirely normal. At the junction of this zone with the white matter, is situated an oval-shaped area of softening which merges into the grey matter, while the neuroglia overgrowth is just beneath the softening (see fig. 1 S^1 S^2, and appears to pervade the entire mass. Surrounding and passing through the softened mass, many diseased blood vessels are seen. The softened area consists of degenerated and disintegrated nervous elements, and a number of bodies which closely resemble Gluge's corpuscles. The neuroglia mass fills up the greater part of the oval area, and is characteristic in every way of this peculiar tissue (see fig. 5).

Ganglion Cells.—Although a large number of the cells appear perfectly normal, many of them show distinct degenerative changes, these degenerative changes are especially seen in the protoplasm of the cells. A number of cells show a more or less granular nucleus, in others the nucleus appears to lie close to the limiting wall of the protoplasmic substance, while still in others no nucleus whatever can be seen. The protoplasmic substance is dotted here and there with empty-looking spaces, which very much resemble vesicles in appearance; in others there is apparently a roughened outline of the walls of the cell. A few cells have

the appearance of a simple wall with a nucleus, but no demonstrable protoplasmic substance. Some of the cells are distinctly larger than normal, probably swollen, while others are considerably smaller and shrunken in appearance. Small bodies resembling a nucleus with its nucleolus, are found scattered here and there amongst normal and degenerated cells. These are unquestionably the remains of degenerated cells, of which nothing remained except the nucleus and the nucleolus. The peri-cellular spaces around some of the cells are larger than they should be, in others the spaces appear well preserved and normal. These changes were present throughout the whole cortex, but were especially distinct in the layer of large pyramidal cells. The whole picture of this cell degeneration is that of a slow, degenerative process, combined with a more or less acute one. Whether an actual hyperplasia of the neuroglia exists in the cortex proper, could not be determined with any degree of certainty, although there were patches here and there which very closely resembled sclerosis.

To recapitulate. The important pathological findings in this case were:—

(1) *Meningo Encephalitis.*—This seems to have been chronic in character; to this there was added an acute exacerbation, which probably occurred in the interval between the first and second operations. Whether or not this chronic meningo-encephalitis had its origin in a trauma cannot be established as fact from what we observe in the specimens under consideration.

(2) *Marked Obliterative Changes in the Blood Vessels of the Pia and Cortex.*—This vascular depravity was associated with the formation of new capillaries which appear to have undergone hyaline degeneration.

(3) *Slow Degenerative Changes in the Ganglion Cells.*—The neuroglia hyperplasia in the cortical areas, although distinctly demonstrable, is not very marked in this tissue. Some acute degeneration was also present.

(4) *Softened Areas.*—These must have resulted from the obliteration of some of the blood vessels, just at the junction of the grey matter with the white, where the blood supply of the brain is poor.

(5) *Replacement of this Softened Area by true Neuroglia Tissue.*—This is well demonstrated in the specimen which shows both softened areas and neuroglia tissue closely associated together in such a way that surrounding each accession of softening there is an advancing zone of neuroglia tissue.

A number of hæmorrhages were noticed in the specimens, but as the blood appeared of comparatively recent occurrence, it is believed that they were produced during the operations.

Case II.

This case, one of idiopathic focal epilepsy, I shall refer to in briefest manner, as the case will probably be reported in detail later by Dr. Dana, to whose clinic she came, and by Dr. Curtis, who did the operation.

The patient, a married woman, 30 years old, of good family and personal history, has suffered for six years from a convulsive affection, which she referred to as a cramp of the left leg, which is often associated with foaming at the mouth, biting of the tongue, involuntary evacuation of excrement, and periods of unconsciousness lasting from five to twenty minutes. The attacks have gradually got more frequent, and now she may have two distinct *hauts mal* attacks and twenty of the cramp conditions of the left leg in twenty-four hours.

Her description of the convulsive attack in the leg and the general seizure in connection with the mental and physical condition after an attack, the increasing frequency of the attacks, and the progressive mental enfeeblement, left no doubt as to the nature of the malady, viz., genuine epilepsy, the attacks of which always began with the same localised spasm.

The cortical area in which the left leg centre is situated was outlined, and a corresponding opening made in the skull. The dura was laid back, and the cortex excised two days later. The piece of brain tissue which was removed measured about 4 cm. in length and 2 cm. in thickness. This tissue was prepared for the reception of the Nissl and picro-carmine stains in the same way as the tissue from the first case.

Sections of the removed cortex prepared in this manner show distinct and, I believe, suggestive changes, particularly in the layer of large pyramidal cells. Without going into histological details, we may recapitulate the pathological findings in this case briefly as follows :—

The shape of the section of the cortex shows that the excised piece was from the bottom of a fissure, and only a small portion of the lateral walls of the fissure are included (fig. 1).

The most striking feature on first examination of the specimens, is the large number of hæmorrhages scattered throughout the entire section, but particularly conspicuous in the deeper parts of the cortex, that is near the line of section. The crimson colour of these hæmorrhagic spots on the cut surface of the specimen indicates distinctly their recent origin. They were, doubtless, due to the trauma incident to the operation. The specimen was extremely hard, and especial precaution was required to cut the sections even with the paraffin imbedding. This excessive hardness was probably due to these hæmorrhages, as nothing else could be found to account for it.

The specimen stained with picro-carmine gives the best general outline of the specimen, and shows the distribution and character of the hæmorrhages (fig. 1). In keeping with the traumatism of the operation the largest patches of hæmorrhage are in the neighbourhood of the line of excision. Towards the surface of the cortex they become smaller, and leave a larger amount of intact tissue between them in the deeper regions. In the superficial layers their size varies from a minute point to a size sufficient to be seen with the naked eye. The larger patches show distinct agglomerations of red blood corpuscles so dense as to almost obscure any other tissue within the affected area. In other places the corpuscles are quite discoloured, and form such a thin and clarified layer that they are hardly seen. In such spots the neuroglia meshes stand out distinctly. In many places only neuroglia meshes are seen. The blood corpuscles and nerve cells have evidently fallen out in this region. This explanation is supported by the fact that all sorts of

transitions of the conditions described are met with. One also sees occasionally a blood vessel within a nearly complete gap of tissue. It is extremely improbable that these gaps are peri-vascular spaces, in view of the fact that in some places blood vessels are to be seen in the middle of patches of completely rarified neuroglia meshes, as well as in the centre of distinctly hæmorrhagic spots.

The multiplicity of hæmorrhages makes it difficult to say whether or not there are distinct vascular and interstitial changes other than those referred to.

Nerve cells. (Figs. 2 and 3).

Specimens prepared after the Nissl method. The superficial layers of cells including the small pyramids down to the layer of large pyramids appear to be perfectly normal. The large pyramids, however, present striking changes. In the first place, there is a great scarcity of these cells (fig. 2). This may be accounted for, in part, by the fact, that at this depth the hæmorrhages occupy a great portion of the tissue, but it is to be remarked that the cells that are found, which by their position and shape correspond to the large pyramids, are for the most part intensely altered, they present the characteristics of atrophy; they are thin, shrunken, with attenuated spiral processes; they stain much more deeply than normal cells, or, at least, they refuse to part with the stain in the differentiation fluid, and they have lost the chromatine striped structure of normal cells. The nuclei which stand out so distinctly in normal cells are here scarcely to be differentiated from the cell body, for it is nearly as dark as the latter. In many cells, indeed, no nuclear structures can be made out. Only a few of the large pyramidal cells met with, present a normal appearance.

The hæmorrhages in these specimens have been described in considerable detail, not because they have, I believe, any particular pathological significance, but to show how numerous and severe hæmorrhages into the substance of the cortex may be after such operations. Particularly in cases where excision of the cortex is done some days subsequently to opening the skull.

It is probable that the blood which formed thin, white,

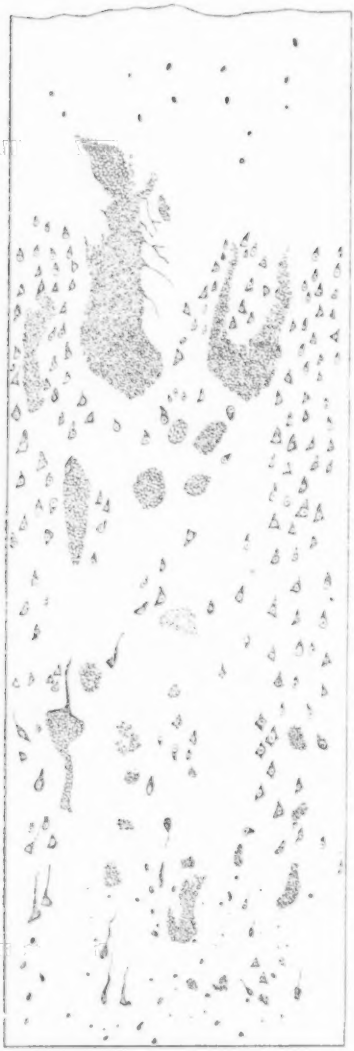


FIG. 2.
Showing scarcity and degeneration of the layer of large pyramidal cells.

FIG. 3.
Individual cells form the layer of large pyramids.

clarified layers dates from the first operation, and the larger more recent looking areas from the second.

The changes in the ganglion cells of the large pyramids we do not believe are secondary to the hæmorrhages, although the scarcity of cells is, undoubtedly, due in part to them. That such striking changes of degeneration could go on in these cells in two days, the time elapsing between the first and second operations, seems scarcely possible.

If these operations should receive corroboration by others, they would indicate not only the direction in which the pathology of epilepsy lies, but the advisability of operation in such cases.

The pathological findings in the first case would seem to be not difficult of interpretation; a patient with Jacksonian epilepsy, has a distinct and localised spasm, which points to a lesion of the cortical centre for the right hand. There are evidences that this lesion has existed sufficiently long to cause beginning secondary changes. The hand centre on the surface of the brain is located and excised. After the acute accompaniments of the operation have passed off, the patient remains free for a year of any attacks; and the only ill results of the operation are the natural sequences, which attend the removal of a part of the motorial area, namely, partial motor disability. Microscopical examination demonstrates that in the piece of cortical tissue which was removed, there are definite pathological conditions which may be postulated to explain the clinical symptoms from which the patient suffered. Logically, the removal of the focus of disease should be followed by a cessation of symptoms, that is, cure of the disease.

The two different lesions that were found in the diseased area may be explained by considering the meningo-encephalitis, in all probability the orginal one. The second set of lesions, the degenerative changes in the ganglionic cells, and the neuroglia-hyperplasia in the cortical areas were secondary to the meningo-encephalitis, and immediately sequential to the vascular obliterative changes in the blood vessels of the pia and the cortex.

The other changes, the hæmorrhages and the diapedesis

immediately about them are probably in no way a part of the patient's disease, but were directly due to the first and second operations.

It is believed, therefore, that the epilepsy in this case was due to the lesions which are herewith demonstrated; that the diseased area in the patient's brain was entirely removed, and that by virtue of such removal the attacks have not recurred; and furthermore by virtue of the very early operative interference, before the motor ganglion cells in the surrounding cortex had become interfered with in their nutrition, or perverted in their function, that the disease will not recur.

We think it advisable not to advance any opinion based on knowledge obtained by the study of the clinical and pathological aspects of these cases, as to what the pathology of so-called idiopathic epilepsy may be, although there is temptation to do so. We are so keenly alive, however, to the liability of error in generalizing from individual cases, that we entirely refrain. We believe, however, that the findings in these cases advance our knowledge of the pathology of epilepsy, insomuch as they show that in the very early stages of that disease there are changes in the sources of motorial impulses, the ganglion cells of the cortex, which can be demonstrated by modern technic.

In closing, it may be said that these cases are not reported as cured, although the first patient has been free from epileptic manifestations for more than a year. The time which has elapsed since operation on the second patient is too short to make statement regarding its efficiency justifiable, although the patient has made gratifying progress since the operation.

If these cases tend to demonstrate anything, it is that there are changes in the brain structure in cases of epilepsy which conscientious surgeons consent to operate on. Therefore it may be asked why should not such diseased tissue be removed in every case? If it be removed, we believe that this operation for epilepsy will not pass into complete desuetude, although it will ever have an extremely narrow field of usefulness.

MIRROR WRITING.

BY F. J. ALLEN, M.D.CANTAB.

Professor of Physiology in Mason College, Birmingham.

A STUDY of the subjective phenomena of left-handed or mirror writing may help to throw light on the nerve processes concerned in writing and other complex co-ordinated actions. The faculty of mirror writing is probably not at all rare. It may be possessed more or less by most persons, but may remain unobserved. It is apt to be discovered for the first time when some nerve disease renders normal writing difficult or impossible; the patient then falls back on the resource of left-handed writing. Thus mirror writing is often a symptom of nerve disease; but the disease need not be the cause of the existence of the faculty, but only the cause of its discovery. At all events in my own person the faculty of mirror writing has existed from childhood. I discovered it accidentally about my thirteenth year, when, on making the first attempt, I was able to write backwards with the left hand without error or hesitation. Ever since that time I have been able to write left-handed without practice.

The most noticeable subjective phenomena are the following:—

(1) It is very easy to write in mirror fashion with chalk on a black board, co-ordination seeming to be perfect in the movements of the left arm and wrist. But when using a pen with the left hand, there is a slight difficulty in some of the movements. There is (in the latter case) no hesitation as to the formation of the letters; but occasional awkward movements will produce distorted lines, and there is special difficulty in keeping up a smooth onward movement, "the movement of alignment."

(2) Writing is only a little slower with the left hand than with the right. In the fastest possible mirror writing there is no error in the form of the letters—only such want of neatness and distinctness as occurs in writing too fast in the ordinary way.

(3) The following actions are quite easy :—

(*a*) Writing the same thing simultaneously with both hands.

(*b*) Writing backwards with the left foot, *i.e.*, scratching on sandy ground, as one sometimes does with the right foot.

(*c*) Using the deaf and dumb finger alphabet in the reversed or left-handed manner.

(4) If I learn a new alphabet with the right hand, I can write it with the left; it is not necessary to learn it separately with the left hand; the nervous system, after being trained to any kind of writing with the right hand, is able to send its co-ordinated messages almost equally well to either hand.

(5) The assistance of sight is not necessary in mirror writing. Indeed, sight may even impede it: and although it is easy to write left-handed, it is difficult to read what one has written. When I look at my own mirror writing, it sometimes conveys no meaning to me, and I experience sensations which are probably much the same as those of a person who is word-blind, but retains the faculty of writing. In order to decipher such writing, it is necessary to run the eye along the lines, and so reproduce the sensations which accompany the act of writing. I find considerable difficulty in reading print reversed by means of a mirror, but hardly any difficulty in reading a book held upside down.

It may be necessary to state that I have no tendency to be left-handed, though I might almost be called ambidexter.

The sensations accompanying all kinds of writing, whether with right hand or left hand, right foot or left foot, are so similar as to suggest that in all cases the messages start from the same region of the brain; but it seems as if a series of commutators existed at a lower level,

whereby the impulses could be turned into different channels leading to analogous but sometimes heteronymous groups of muscles. This supports the view that the true graphic centre is not coincident with either of the motor centres, but superior to all of them.

I append some words written both in mirror and in ordinary writing; the features may be compared with the assistance of a mirror.

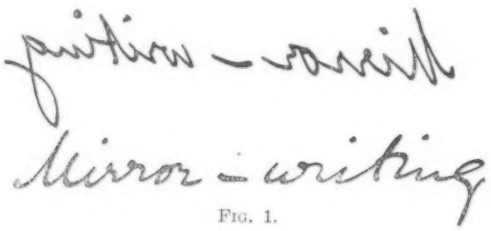


FIG. 1.

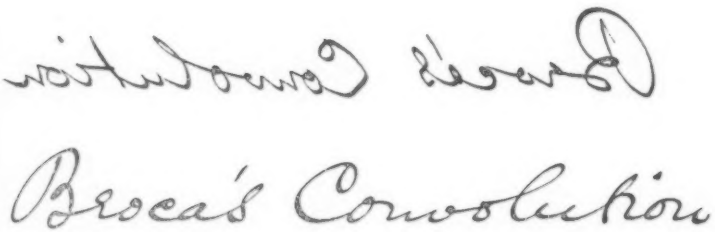


FIG. 2.

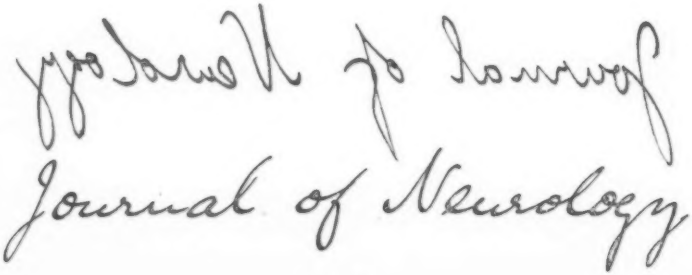


FIG. 3.

Critical Digest.

ABSTRACTS OF PAPERS ON THE ETIOLOGY AND PATHOLOGY OF DISSEMINATED SCLEROSIS.

Etiology.

Marie (1), in discussing the etiology of the disease, points out that the most common age at which the symptoms commence is the early part of adult life (20-30), and he regards this as a point of some importance in the diagnosis. In any case of doubtful diagnosis, disseminated sclerosis may be almost eliminated, if the disease has commenced after the 40th year. On the other hand, however, the disease is occasionally met with in infants. Marie believes that infectious diseases are the most important factors in the causation of disseminated sclerosis. When one considers the number of facts in favour of this relationship published by various authors, and the number of cases in which the affection of the nervous system has first shown itself in the course of the infectious disease or during convalescence therefrom, Marie thinks it is impossible to deny that there exists a relation of cause and effect.

As regards the nature of these infectious diseases, Marie found, in twenty-five cases which he collected, that typhoid fever was the antecedent in eleven. Pneumonia, malaria, measles, scarlet fever, and especially small-pox, have been followed by disseminated sclerosis. Other infectious diseases—diphtheria, whooping cough, erysipelas, dysentery, and cholera—have occasionally been recorded as antecedent in disseminated sclerosis. To this list Marie adds "unknown infectious diseases." He discusses the relation of disseminated sclerosis to the micro-organisms of infectious diseases. If these micro-organisms were the cause, one would expect to meet with disseminated sclerosis much more frequently in the course of various infectious diseases. Hence he regards a combined or mixed infection, which is so frequent in the course of the infectious diseases as the exciting cause. Though it is not possible, at present, to come to any definite conclusion as to the exact pathogenesis, still Marie thinks it is a well-established fact, that disseminated sclerosis stands in close relation, as regards causation, with the infectious diseases.

Totzke(2), in his inaugural dissertation on disseminated sclerosis in childhood, quotes cases, published by Leuch, of a mother and child, who both suffered from the disease. The mother was suffering from the disease during her pregnancy; the child was healthy until the 7th year; then symptoms of disseminated sclerosis appeared, and the disease terminated fatally. From a review of 35 cases in children, 33 of which were collected from medical literature, Totzke regards infectious diseases as the most important factor in the etiology. In these 35 cases the disease followed some acute infectious ailment in 13—diphtheria, 4; scarlet fever, 4; measles, 1; whooping-cough, 2; cholera nostras, 1; unknown febrile affection, 1. The author mentions two instances, in which two children of the same parents suffered from disseminated sclerosis.

In 31 cases of the disease in childhood, the onset was before the 6th year of age in 19 cases; in 12, between the ages of 6 and 14 years. In 2 cases the symptoms appear to have been present from birth. In disseminated sclerosis in the adult, the commencement of the disease can sometimes be traced to childhood. In some cases the patient has presented typical symptoms in childhood; in other cases certain symptoms only have been present.

A case of disseminated sclerosis following influenza is reported by Massalongo (3). The writer has met with a case also, in which the disease followed influenza. But considering the prevalence of influenza during the last few years, more evidence is required before any conclusion can be drawn with regard to the connection of the two affections.

Torti and Angelini (4) record two cases of chronic malaria, in which symptoms resembling those of disseminated sclerosis developed. The first patient was a man aged 21 years, who, after suffering for three months from malarial fever of an irregular type, began to be troubled with vertigo and vomiting. Then weakness of the legs, slow scanning speech, increase of the knee-jerks, slight nystagmus, were noted. Examination of the blood revealed the malarial parasites. Under treatment, improvement occurred, and the malarial parasites disappeared from the blood. The patient was discharged from the hospital, but in a short time a return of fever occurred, and in a few days the following symptoms developed: spastic paresis, ataxia of the limbs, nystagmus, tremor, increase of the reflexes, scanning speech, headache, and vertigo. The malarial parasites were again found in the blood. The symptoms again subsided, and in two months

the patient was discharged well. The second case was a man aged 22. In August and September, 1890, he had suffered from malaria. In November he presented the following symptoms: headache, vertigo, scanning speech, nystagmus, intention tremor, increase of the reflexes. The malarial parasites were present in the blood. After three weeks' treatment he recovered.

Cases of multiple sclerosis after malaria have been recorded by Camellis, Boinet and Salebert (pseudo-sclerosis), and Bignamic Bastianelli. Clinically, three forms of sclerosis are met with after malaria. (1) Symptoms of multiple sclerosis may occur temporarily with severe attacks of malarial fever. (2) Symptoms of multiple sclerosis (the duration of which varies) may occur after the fever. (3) Symptoms of sclerosis may occur suddenly with fever under the form of very acute malarial attacks. All three forms show a tendency to improvement and recovery. Torti and Angelini believe that the cause of these symptoms is a poisoning of the nerve centres (not a vascular disease), and refer to a case of pernicious malaria, recorded by Marchiafara, with bulbar symptoms (paralysis of the facial, hypo-glossal and vagus) in which the vessels in the region of the bulbar nuclei were filled with blood cells containing parasites (parasitic thrombosis).

Jordan (5) reports a case, under the care of Suckling, in which symptoms of disseminated sclerosis came on suddenly a few days after a sudden and violent mental shock.

Leyden (6) states that cases, beginning like an acute myelitis, sometimes pass on to sclerosis. Disseminated myelitis may follow an acute illness and *post-mortem* examination, after the nervous symptoms have existed for many years, may reveal multiple sclerosis.

In many cases of spinal paralysis which present the symptoms of acute myelitis, the autopsy made after the disease has existed for many years, has revealed sclerosis of the spinal cord.

Pathology.

Charcot, in his earlier work on the pathological anatomy of disseminated sclerosis, drew attention to the dilatation of the blood vessels in diseased patches and to the thickening of their walls, especially of the external coat. Charcot's view as to the interstitial origin of the sclerosis has been accepted by most pathologists, but not by all.

Huber (7) has quite recently reported a case of disseminated sclerosis, in which, in some parts, the diseased patches presented

simply disappearance of nerve fibres without changes in the interstitial tissue, or without changes sufficient to cause the destruction of the nerve fibres. Also in the patches the vessels were not changed, and showed no relation to the distribution of the diseased process. In these patches the author regards the disease as a primary affection of the parenchyma. In other patches there was marked sclerosis of the interstitial tissue, and between the two kinds of patches there was no sharp distinction. Hence the author believes that the patches belong to one diseased process, in which an affection of the parenchyma is the primary change, and the sclerosis secondary. Further, in longitudinal sections of sclerotic patches, sometimes small linear processes were seen, which were situated exactly in the course of the atrophied nerve fibres, connective tissue taking the place of degenerated nerve fibres. If the changes proceeded primarily from the interstitial tissue, one would expect the new formed neuroglia to be equally developed laterally at these parts.

The vascular origin of the patches in disseminated sclerosis was suggested by Rindfleish many years ago. More recently, Ribbert (8) from a study of the microscopical changes, has arrived at the conclusion that disseminated sclerosis is related to vascular lesions, and due to the presence of some irritating agent in the vessels. In the case which he reports, both greyish-red, soft, recent patches, and old grey patches, were found at the autopsy. In the former, a large, greatly distended blood vessel was found running through the centre of each. Around the vessels in these patches there was a collection of round cells. In two places he found partial obstruction of the lumen of an artery by a peripheral thrombus, consisting of white blood corpuscles.

Ribbert believes that some irritating substance is distributed by the vessels to the central nervous system. Under the influence of this irritating agent a fibrinous clot forms at one part of a small vessel. The clot generally occupies only a portion of the periphery of the lumen of the vessel. At this point an irritation of the vessel wall occurs, and more or less marked diapedesis of lymph corpuscles follows. A peri-vascular inflammation occurs, which extends in a concentric manner around the vessel from which it commenced. The exact nature of this irritating agent remains unknown.

Bastian (9) states that on several occasions he has found the larger vessels of a patch of sclerosis blocked by old and firm thrombi.

Marie (10) draws attention to the vascular changes in dis-

seminated sclerosis. In the centre of a patch of sclerosis a dilated blood vessel is often found, and the sclerotic patch appears to have developed around the vessel. Considering the relation of disseminated sclerosis to infectious diseases this localisation is not surprising. Marie points out two interesting facts with regard to the patches of sclerosis. One is, that the periphery of many patches presents more or less numerous granular corpuscles, which indicate that the morbid process is still active. A second interesting fact is that in some old standing cases, at the autopsy, in addition to old patches of sclerosis, there are small patches containing granular corpuscles both at the centre and the periphery—in other words, recent patches of disease. The first fact above mentioned indicates that the morbid agent remains in the patch and continues its action in an almost indefinite manner. The second fact shows that the morbid agent persisting in the organism is able to diffuse itself and to cause the development of new patches of sclerosis. Though our definite knowledge as to the causation of disseminated sclerosis is so slight, Marie thinks, however, we may accept the following conclusions: disseminated sclerosis is a sclerosis of vascular origin, the general aspect of which appears to indicate a process allied to *embolism:* it arises under the influence of various infectious diseases, probably by the mechanism of combined infection.

Hess (11) describes the pathological changes in a case of disseminated sclerosis, drawing attention to the persistence of axis-cylinder in the sclerotic patches and to the absence of secondary degeneration. In addition to the patches of firm sclerosis, both in the medulla and cord, areas of diffuse neuroglia proliferation were met with, in which the medullary sheaths of the nerve fibres were simply diminished and not absent. The condition of the vessels varied in different parts. Many small vessels, especially the capillaries, were normal. At some parts the walls were thickened, chiefly in the larger vessels; or the vessels were increased in number; or they were dilated; in other parts the lumen was diminished. At some parts the lumen was completely obliterated by thickening of the vessel walls. The peri-vascular lymph sheaths were distended with round cells and the nuclei of the vessel walls increased.

In addition to the patches presenting the usual appearances of disseminated sclerosis, there were in the pons, medulla, and at one part of the lumbar region of the cord, areas in which there was marked cell infiltration. These patches the author regards as the results of circulatory changes and as an early stage in the development of disseminated sclerosis.

Hess believes that the sclerosed patches commence with marked disease of the vessel walls. In the patches of diffuse sclerosis, he believes that the nutrient vessels are only so far diseased that an emigration of white blood corpuscles and an increase and new formation of the neuroglia occurs, analogous to the increased interstitial tissue in the disturbance of nutrition in other organs. Hence only here and there does the myelin of the nerve fibres disappear. In other vessels supplying sclerosed areas as the changes in the walls are more marked. As a result the neuroglia is increased and the white substance of the nerve fibres disappears owing to defective nutrition. Only after a long period of disturbance of nutrition does the axis-cylinder disappear. The changes in the vessel vary in degree. They are, at some parts, only slight and allow the emigration of white corpuscles to occur. In other places the changes are marked and may lead to complete obliteration with severe nutritional disturbances in the region of distribution; between both these extremes there are various modifications.

By obliteration of the vessels (owing to thickening of the walls) the same circulatory disturbances would be produced as by the peripheral thrombi observed by Ribbert. The author looks upon disseminated sclerosis as a process in which the myelin of the nerve fibres disappears and the neuroglia proliferates owing to primary changes in the vessels.

Popoff (12), from the microscopical examination of the central nervous system in four cases, comes to the conclusion that the usually accepted views as to the pathology of disseminated sclerosis are erroneous. He recognises two forms of the disease —sub-acute and chronic. He does not believe that there is an increase of the connective tissue in the sclerosed patches, but thinks that what most observers have looked upon as tracts of connective tissue, lying between the nerve fibres, are only degeneration products of the nerve fibres themselves. He regards the methods of staining, which have been usually employed, as unsuitable for studying the nature of the tissue which replaces the nerve fibres in the sclerosed patches. The vessels, which form the centre of every patch, must be looked upon as the starting point of the affection. The changes in the surrounding tissue spread gradually from the central vessel, and this gradual advance of the process is especially marked in the chronic cases. All the tissues around the vessels are finally affected, but not at the same time.

The chief change in the vessel wall consists in cellular infil-

tration, followed by thickening, and sometimes concentric narrowing of the lumen. Also an abundant emigration of leucocytes occurs into the surrounding tissue. The leucocytes do not increase in size; they do not become changed into connective tissue, but degeneration. Through their presence between the nerve fibres probably they exert an injurious influence on the vitality of the fibres, but they are not the chief cause of the metamorphosis.

The sub-acute form of multiple sclerosis differs from the chronic in this, that in the former the cell infiltration of the vessel walls is more irregular than in the chronic form; the number of leucocytes is distinctly greater, and the course is more acute. In some place the author has found what he regards as regenerated axis-cylinders, and he believes that the great numbers of axis-cylinders without medullary sheaths, which have been described by Charcot, Leuker, Hess, and others, in sclerosed patches, are to be regarded as new-formed axis-cylinders—regenerated axis-cylinders.

At the periphery of a sclerosed patch a new formation of blood vessels occurs—a condition which the author regards as favourable to the regeneration of axis-cylinders of nerve fibres.

According to Popoff, the generally accepted views as to the pathology are false, because—

(1) No proliferation of connective tissue occurs in this disease.

(2) Not only do the nerve fibres lose their medullary sheaths, but the axis-cylinders also degenerate.

The pathological process consists in a gradual degeneration of the whole tissue surrounding an affected vessel. Besides the degenerative process, a regeneration of axis-cylinders occurs, perhaps only in certain parts of the cord (the pyramidal tracts and Goll's columns).

Buss (14) reports a case which during life presented symptoms of spastic spinal paralysis, but the autopsy revealed disseminated sclerosis, with secondary degeneration (ascending and descending) in the spinal cord. In many places the axis-cylinders were destroyed. Peri-vascular cell infiltration was marked, and the walls of the vessels were thickened. In the brain there were numerous small patches; in the centre of the patches a small vessel was seen on section. These vessels were veins with thickened walls, which were surrounded by cell infiltration. Buss regards the vascular changes as the starting point of the lesion.

The clinical history of the case is unusual, from the fact

that there were no tremors on voluntary movement and no scanning speech. The first symptom was dull pain in the back. A few weeks later, after severe exposure to cold and wet, the patient suffered from a marked rigor, accompanied by vomiting and pains and tremors in the limbs. After this attack the nervous symptoms became much worse. The same acute symptoms followed a large dose of aloes (forty-one pills taken one after the other), and afterwards the spinal symptoms again rapidly became worse. Whilst the patient was in the clinic these symptoms —marked rigors with high temperature, &c.—occurred several times. In the attacks the urine became dark, and contained blood pigment. After one of these attacks of rigor, with febrile symptoms and hæmoglobinuria, the spinal symptoms for a third time became rapidly worse, and an acute bed-sore developed. Buss believes that the rapid advance of spinal symptoms, after the large doses of aloes, was due to blood dissolution. Aloes, in toxic doses, may give rise to necrosis of the renal epithelium and hæmoglobinuria. Small doses of substances producing hæmoglobinuria have been found to produce thrombosis in the small vessels of the heart, the lungs, and in the small veins in other organs. Buss found in his case of disseminated sclerosis that the sclerotic patches were grouped, almost without exception, around small veins, but no thrombi were detected.

Buss thinks that in several points there is a strong resemblance between an attack of hæmoglobinuria and the effect of exposure to cold. There are several facts which render it probable that the acute symptoms following the exposure to cold and wet in the above-mentioned case were due to hæmoglobinuria. The author believes that (1) after the exposure to cold; (2) after the aloes pills; (3) after the attacks of hæmoglobinuria, similar general blood conditions occurred; on all these occasions the spinal symptoms suddenly advanced markedly.

Afariassiew and others have shown that in hæmoglobinuria, produced experimentally, interstitial changes are found in the various organs. They attribute the changes to the irritating (or inflammation producing) properties of substances formed by blood dissolution. Buss believes that in consequence of blood changes (dissolution) some substance is produced which has an inflammatory exciting influence on the small veins of the nervous system. Changes are produced in the walls of the veins, and secondary changes occur in the nervous system around. This he looks upon as one cause only of disseminated sclerosis. Many patients refer the disease to a severe cold (taking cold),

and Buss thinks that blood changes may be produced thereby, and a hæmoglobinæmia produced without hæmoglobinuria, and this may be an explanation of the apparent influence of cold in producing the disease in some cases.

Taylor (15) gives a detailed account of the pathological changes in three cases of disseminated sclerosis. In the first case, the whole cross section of the spinal cord at many regions was degenerated. Often only a small number of fibres which still retained their white substance, were recognised on a transverse section. Both grey and white substance were affected indifferently. The extra medullary parts of the anterior and posterior roots were degenerated at some parts. The nuclei of all the cranial nerves (in the medulla, pons, and region of the corpora quadrigemina) were affected. The central canal of the lower part of the medulla, the whole floor of the fourth ventricle, and the aqueduct of Sylvius up to the oculo-motor region were involved in one continuous patch of sclerosis.

In the second case, it is worthy of note that the cauda equina, the spinal roots, and the cortex of the cerebellum were affected: and in the third case, the internal capsule, cortex of the cerebrum and cerebellum were affected.

In opposition to the views of previous writers, Taylor concludes from his own observations that the white and grey substances are affected irregularly; that there is no prædilection spot in the central nervous system for the patches of sclerosis; that neither the cortex of the cerebrum nor cerebellum are spared.

In two of Taylor's cases all the cerebral nerves were affected (except the olfactory) at some part; either at the nucleus, or in the central or peripheral course of the nerve.

The optic chiasma was also the seat of sclerosis in all of Taylor's cases.

The author confirms the observations of Charcot and Erb, that the spinal nerve roots are often affected; he also found well-marked degeneration of the nerve fibres of the cauda equina (as above mentioned).

According to Charcot, at the periphery of the patches of sclerosis there is a transitional zone, of less marked degeneration before the healthy nerve tissue is reached. But Taylor has observed many sclerosed patches, which were sharply defined, and in which there was no transitional zone of partial degeneration, a patch of marked sclerosis, in which medullated nerve fibres were absent, being bounded by healthy nerve tissue.

Taylor has also found complete atrophy of the medullary sheaths of the nerve fibres, both at the centre and at the periphery of the patches. In some patches the whole area affected, on microscopical examination, presented the appearance which has been described as that of the terminal stage.

The author refers to the absence of secondary degeneration in disseminated sclerosis, which has been so frequently noted; but he admits the possibility of such changes when the axis-cylinders are completely destroyed in a sclerosed patch, though he believes a well-marked secondary degeneration of the various tracts to be exceedingly rare.

Taylor's observations show that degeneration of ganglion cells only occurs in an advanced stage of the process, and not directly the grey anterior horn is implicated, as is stated by some observers. Undoubtedly the cells persist for a long time, and, for the most part, are in a state of functional activity. This is the reason why the reaction of degeneration and muscular atrophy are so rare in this disease. The first change in the cells consists in marked pigmentation. In the normal spinal cord pigmentation is observed, but not to such an extent, and in such young individuals, as in disseminated sclerosis. A collection of pigment which fills the whole cells does not occur under normal conditions. A further degeneration, up to complete atrophy of the cell, only rarely occurs, and that in the last stages of the disease.

The changes in the vessels in Taylor's cases were not marked. In one case the small vessels, especially the capillaries, were enormously increased in some regions. They were filled with blood; here and there were small hæmorrhages, and often extravasated white blood corpuscles. Some vessels showed moderate thickening of the walls, with a minimal increase of nuclei. The peri-vascular spaces were often dilated.

Similar changes, but less marked, were found in the second case. In the third case, scarcely any abnormality of the vessels could be detected. In the first two cases, the vascular changes were most marked at the floor of the fourth ventricle, where the sclerotic degeneration was very extensive. Taylor, however, is of opinion that the vascular changes were not the starting point of the sclerosis for the following reasons:—

(1) Because the patches were not always distributed in relation to the vessels.

(2) Because in markedly degenerated parts, often the vessels showed no essential changes.

(3) Because in one case vascular changes were absent.

[It is to be remembered, however, that in Taylor's cases the pathological examinations were made at a comparatively late stage of the disease.]

A case reported by the writer (16) illustrates several interesting points with regard to the pathology of disseminated sclerosis. The course of the disease was very rapid. Indefinite symptoms were noticed twelve months before death, but distinct symptoms of the disease only appeared within the last five months of life. On pathological examination, in addition to old disseminated patches in the brain and cord, presenting the usual naked eye and microscopical appearances of multiple sclerosis, there were four *very soft* cerebral patches. The largest was in the right temporo-sphenoidal lobe ; it was greyish-yellow in colour, almost diffluent, and from the cut surface a small quantity of turbid greyish fluid escaped. There were other cerebral patches intermediate in consistence between these soft cerebral patches and the firm sclerosis patches in the cord. Sections of the softened patches, even at the centre, were seen to consist almost entirely of round cells, compound fat granular cells, and dilated blood vessels, the peri-vascular sheaths of which were distended with leucocytes. The appearance closely resembled that of acute cerebral softening. In the firmer patches there was increased fibrous connective tissue, and the usual microscopical appearance of sclerosis. A striking feature in nearly all of the patches, was the marked dilatation of the blood vessels, and the distension of the lymph sheath with leucocytes. In the upper cervical region was a recent wedge-shaped patch, in the region of the anterior median fissure. This area was densely infiltrated with leucocytes. The vessels in the fissure were greatly dilated and the peri-vascular lymph sheaths distended with leucocytes.

At the commencement of the anterior median fissure (*i.e.*, near the surface of the cord) in a number of sections there was a thrombosed vessel. In some of the sections taken from this part of the cord, in addition to the thrombus in the anterior median vessel, there were several small thrombosed veins in the pia mater on the anterior surface of the cord.

These thrombi are of interest in connection with the above-mentioned views of Ribbert and Marie, and the observations of Bastian and Ribbert.

Demange (17) reports an interesting case of sclerosis of the cord of vascular origin. During life the symptoms resembled those of amyotrophic lateral sclerosis. At the autopsy extensive athe-

roma of the vessels was found. The microscopical examination revealed disseminated sclerosis in all regions of the spinal cord. These patches were scattered most irregularly in the white matter, and in some parts penetrated the grey substance. The margins of the patches were not sharply defined, but were furnished with radiating processes (sclérose "non en plaques mais en taches"). In the centre of each small area of sclerosis was an altered artery, from which the sclerosis extended into the meshes of the neuroglia. The neuroglia was thickened and possessed numerous spider cells. The arteries appeared generally increased in number, and of unusual size. The endothelium was proliferated, the walls thickened, infiltrated with nuclei, and the lymph spaces filled with leucocytes. Demange regards the case as one commencing in a diffuse myelitis, proceeding from the peri-arteritis of the vessels—an interstitial, diffuse, and peri-vascular myelitis. He divides scleroses of vascular origin into two varieties (1) the disseminated sclerosis in patches; (2) certain interstitial diffuse scleroses, some cases being due to syphilis, others being connected with general atheroma of the vessels.

Demange (18) also reports two other cases with similar arterial changes. From the changes in those three cases he concludes: That the vessels of the cord may be affected by a peri-arterial sclerosis, connected with general atheroma. This lesion gives rise to spots of diffuse sclerosis (of vascular origin) and also to miliary hæmorrhages. Clinically the symptoms are those of sclerosis of the lateral columns of the cord.

Déjérine (19) thinks that it is more than probable, that the non-symmetrical scleroses of the spinal cord are all of vascular origin.

Hence, though the pathology of the disseminated sclerosis is as yet very obscure, a number of interesting points have, nevertheless, been made out.

(1) The distribution of the sclerosed patches in the most irregular manner throughout the nervous system, without any relation to nerve tracts or other nervous structures, whilst adjacent parts of the nervous system are normal, favours the view that the sclerosed patches are probably related to the distribution and lesions of the blood vessels.

(2) It is probable that some cases of disseminated sclerosis commence as myelitis or encephalitis.

(3) At the periphery of many patches of sclerosis, more or less numerous compound granular cells are found, with indications that the morbid process is still active. Also, in some cases at

the autopsy, in addition to old firm patches of sclerosis, there are soft patches containing compound granular cells both at the centre and at the periphery, *i.e.*, patches of recent development. These cases show that the morbid agent persists in the organism and is able to diffuse itself, and cause the development of new patches of the disease.

(4) Numerous observers have pointed out that in the earlier stages of the development a small artery is found in the centre of each sclerotic patch, and the chief changes are found just around the vessel.

(5) In a large number of cases distinct vascular and peri-vascular changes have been found, especially in the early stages of the development of a patch of sclerosis. In several cases thrombi have been found in a few vessels. These changes are very suggestive that the disease is associated with vascular lesions, either in the nature of a partial or complete vascular obstruction (multiple thrombosis of minute vessels), or of a process *allied* to multiple embolism (Ribbert and Marie), or possibly the presence of some irritating agent in the circulation may stimulate the endothelium cells of the walls of the vessels, and give rise to extravasation of lymph cells, and peri-vascular changes at certain spots, and as a result patches of degeneration may follow.

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Reviews and Abstracts.

Studien über Hysterie. Von Dr. Jos. Breuer und Dr. Sigm. Freud, in Wein. Leipzig und Wein. Franz Deuticke, 1895, pp. 268.

These "Studies" form an original and valuable contribution to the theory of hysteria. The authors, in 1893 and 1894, published short preliminary communications of their observations on hysteria, and they now give in this book a full statement of their views. In Brain, vol. xii., pp. 125, *seq.*, will be found an abstract of their first papers. Briefly they pointed out in these papers the importance of ascertaining the exciting cause of hysterical phenomena, and the circumstances attending their first appearance; the influence in producing hysteria of "psychical" shock, and of violent emotional disturbances, for which there is no outlet: that on a "reaction," or outlet, for these pent-up emotions being afforded through the agency of speech, relief to the symptoms could often be given; and they described the existence of "hypnoid states" in hysteria.

In the former communications there was no room for cases, but in this volume five selected cases are given at length in order to show their method of procedure, and the nature of the evidence on which their views are based.

The essential particulars in the analysis of one case by Dr. Freud may be given. The patient, a young lady, aged 21, had suffered during the previous three years from family troubles; she had nursed her father through a long and painful illness, which ended fatally; then she nursed her mother through a severe ocular affection, necessitating a long stay in darkened rooms; and after that she lost a favourite sister, who died somewhat suddenly after a short illness. She complained of difficulty in walking, on account of pains, which were increased by walking. There was some tenderness of the skin and muscles of both legs, and an imperfect hysterogenic zone on the outer aspect of the right thigh. The history of common emotional disturbance afforded no explanation

why the patient should suffer from hysteria, nor why it should take the form of pain on walking and on movement of the legs. The first origin of the pain possibly originated whilst she was nursing her father, from jumping out of bed with bare feet to attend upon him. At this time she had also a love affair, and there was a conflict between her inclinations and her duty to her father. The hysterogenic zone on the thigh corresponded in position to the spot on which she supported her father whilst the dressings were being changed. Further examination under partial hypnosis elicited the fact that pain on walking had been first felt after a long walk with her sister's husband, during which she discovered that she regarded him with more tender feelings than she should do. Moreover, pains in walking, standing, or lying down seemed to be connected in memory with painful scenes in which she had been standing, walking, or lying respectively. The first period of her illness could be described as a monosymptomatic hysteria, *i.e.*, pains in one thigh connected with the strain of nursing her father; the second period in which the pains spread to the other leg originated in the shock of her sister's death. (See below.)

During the first period of treatment, after the above facts had been ascertained, she improved considerably. Variations in her condition could be traced to definite causes, such as the account of a friend's illness recalling to her mind her father's death, or the sight of a child reminding her of her dead sister's child. The author thought that the painful area on the thigh increased in size with the accession of fresh "psychical traumas." As after a time the patient made no further improvement, she was again put under the authors' "cathartic method" of treatment. It was then elicited that, as above mentioned, she had fallen in love with her brother-in-law. The news of her sister's illness had reached her when she was living at a considerable distance; and after a long and tedious journey in anxious suspense, she arrived only to learn that her sister was dead. She was horrified to find that the first thought that arose involuntarily to her mind, and against which her whole moral being revolted, was that her brother-in-law was now free to marry her. Then occurred the "conversion" of psychical into somatic innervations; this idea, so repugnant to her moral sense, being directed into somatic channels, and becoming the origin of fresh hysterical symptoms. The author then put clearly to his patient what he had gathered from her more or less rambling statements. This threw her into great agitation, and she denied what she had said. It was shown

her that no other interpretation could be placed on her own recitals; and subsequent interviews with her mother confirmed the accuracy of the main facts. The mother further said that no realisation was to be expected of the patient's wish to marry her brother-in-law. The subsequent history of the case was that after a period of illness she completely recovered her health.

Dr. Freud, commenting on the case, remarks that there was no evidence of hereditary tendency to nervous disease. The germs of hysteria were developed during the long period of strain and anxiety when the patient was nursing her father; his illness prevented her from paying attention to her own symptoms—"*retentions hysterie*"; had he recovered there would have occurred a rebound to a normal state of health; as he died, all the factors present necessarily led, after a short interval, to an outbreak of hysteria. The onset of pains in the thigh coincided with a conflict between her duty to her sick father and her erotic inclinations. The erotic ideas were forced out of consciousness, but replaced by physical pains. This conflict would probably take place many times over, and its consequences be thereby accentuated. Later, a conflict of the same kind arose between her love for her brother-in-law, both during her sister's life and after her death, and her moral feelings, followed by the development of fresh pains in the course of a period of exhaustion, the result of a fresh bout of nursing during her mother's long illness. Her love for her brother-in-law does not seem to have been consciously clearly realised, but remained as a "foreign body," apart from the other contents of consciousness, and not subject to associative interchange.

In their preliminary communication the authors were careful to speak of "the psychical origin of hysterical phenomena" rather than "of hysteria," because they do not wish to claim for their view an unlimited importance. They neither believe that all hysterical phenomena arise in the above described way, nor do they agree with Möbius that they are all dependent upon idea. They hold hysteria to be a form of disease discovered empirically from clinical observation, and the name a convenient clinical term like pulmonary phthisis. The knowledge thus empirically gained has been extended and simplified, but not destroyed, by the growth of knowledge. Though research has shown that pulmonary phthisis comprises different diseased processes, excited by the tubercle bacillus and by the micro-organisms of suppuration, &c., yet the term is rightly preserved as a clinical entity. So the term hysteria must remain; although

further knowledge may establish that its phenomena are due to different causes, some of psychical, others of non-psychical nature. Möbius's definition that "all those morbid symptoms are hysterical which arise from idea" is objectionable, because it breaks up hysteria as a clinical group of symptoms.

It seems certain that many hysterical phenomena do not arise through idea. Dr. Breuer takes as an instance hysterical pains; he admits that some of these do often arise directly from idea. But besides great vivacity of representation, abnormal sensitiveness of the pain-conducting and pain-receiving apparatus seems to be required. Though the expression, pain-hallucinations, vividly expresses the nature of these neuralgias, the ideas or memories concerned are never sufficiently intense to have that character of objective existence which marks an hallucination. In healthy persons memories of bodily pains cannot be called up with the same vividness as visual and auditory memories; in deep sleep pain is not dreamt of unless some real sensation of pain is present. He postulates, therefore, an abnormal irritability of the sensory centres for pain to explain the readiness with which "pain hallucinations" occur in the hysterical. In an arthralgia, arising from slight injury to a joint, a psychical element is present, but the affection can scarcely be said to originate through idea. So with pathological lowering of sensation. It is improbable that general or local analgesia and anæsthesia are produced through idea; and if Binet's and Janet's views are confirmed that hemianæsthesia is connected with a peculiar psychical state—*i.e.*, narrowing of consciousness—it is psychogenic, but not "ideogenic"; and, according to Möbius's definition, is not hysterical. Dr. Breuer would, therefore, say that many hysterical phenomena, probably more than we are at present aware of, originate through idea; but the fundamental morbid change is an abnormal irritability of the nervous system; and it is a further question as to how far this is of psychical origin.

Passing on to the question of what constitutes a "nervous" person, the author argues as follows:—Sleep and waking form the two extreme conditions of the nervous centres; sleep differs from waking in that in the former the association and conduction-paths in the brain for the excitation of the psychical elements (? cortical cells) are blocked. He further supposes with Exner that during waking there is a state of intra-cellular tension, that the conduction-paths are in a condition of tonic excitation, and that this intra-cerebral excitation is bound up with the power of conduction. In a man expectant and ready for some mental

or physical work, the tension or irritability of the conducting paths in the brain is increased. This involves wear and tear, and cannot be indefinitely maintained. Sleep means the restitution of brain energy, and unlike muscle, which only enters in action when stimulated, the brain begins to act when restitution is complete. If the brain remains at rest too long—no demand being made on it for mental or bodily work—a desire or necessity for activity is aroused, which, if not carried into effect, becomes painful. There is, therefore, postulated a *tendency to maintenance at a constant state of the intra-cerebral irritability*. If a proper means for its discharge be not found, and it is in excessive amount, the excess passes off in purposeless motor activities. The maintenance of the above-mentioned condition of strain or expectancy at appropriate times is essential to the performance of the best work; but if constantly recurring on inadequate or unsuitable occasions, it is hurtful; and is the characteristic state of the *nervous* individual. Certain organic sensations connected with the primary needs of the organism, *e.g.*, need of oxygen, are bound up with special mechanisms co-ordinated for their satisfaction, and are further dependent on chemical changes in the constitution of brain elements. The sexual functions, with their imperative need for satisfaction of the sexual sense, form a transition stage between the above elementary sensations and the psychical emotions. Emotion disturbs the dynamical equilibrium of the central nervous system, and causes an increased excitability. This increased excitability is discharged by means of the motor activities bound up with the emotion. Thus, in anger, the resulting wrathful actions lessen and relieve the increased cerebral activity. Emotions, such as terror or anguish, have, however, a depressing effect, and the disturbance they cause in the central nervous system only disappears through a gradual adjustment. If no such motor outlet is afforded to the excessive intra-cerebral irritability thus arising, the disturbance may still gradually disappear in healthy men; but in many persons abnormal reactions will occur, that is to say, there is an abnormal discharge of emotional movements.

Next there is a tendency in hysteria to change psychical innervations into physical (bodily movements). The motor activities by which relief is normally given to the increased intra-cerebral irritability produced by the emotions, are fixed and co-ordinated movements; but an excessive irritation may break through the usual paths and stream out in elementary movements; these are opisthotonus, representing the maximum effort of all the muscles,

and kicking and struggling. Such abnormal reactions to emotion are not confined to hysteria, and are only to be considered hysterical if they occur spontaneously without the incitement of an objectively grounded emotion.

Similarly with regard to the revival in memory of such past emotions. If at the time that an injury or insult is received, the recipient is able to freely express his indignation; subsequent recall of the injury in memory is attended with little excitement or irritation. Should, however, he have been unable to express his anger, recall of the event is associated in memory with the angry words, and forms the "psychical reflex" of the state of irritability. If the original emotion has been discharged by an abnormal motor action, the latter will also be recalled; in other words, the irritation arising from the recall of the emotion bound up with the conception of the injury is changed or converted into a physical innervation. Frequent repetition may so firmly establish this reflex act that the emotional element drops out; and, finally, as conceptions, unaccompanied by emotion, are far less vividly recalled in memory, the concept may also disappear, and the change into a motor act be perfect. The intra-cerebral irritation of the emotion has been diverted into a process of excitation of peripheral paths; and the concept, originally of emotional origin, recalls now, not the emotion but the abnormal reflex act. Further, the occurrence of the particular reflex act which may be present in any individual case, will often be determined by some emotion or voluntary action concomitant at the time of the original disturbance (path of least resistance).

Strong sensory impressions concomitant with the original emotion ("affecte") may accompany its revival in memory, not as a remembrance, but as an hallucination. In the higher grades of hysteria there often exists, between the original emotional disturbance and its (motor) reflex act, an active group of associated concepts which the author names "*Determinirung durch Symbolik.*" In very many cases these hallucinations are incomprehensible, because the key to their origin is unknown to us. The disturbances (experience) which set free the original emotion whose irritation has become changed into physical innervation, the author calls a "psychical trauma," and thus assimilates cases of hysteria originating in this way to those of traumatic origin in the ordinary sense.

The psychical causes of pathological (hysterical) manifestations are religious doubts, conflict between evil inclinations and the moral sense, inability to follow in the path of duty, and the

sexual feelings and impulses which are especially potent. The pathological change may be in the disposition or temper (as in the conditions termed *Angstzustände*), or in the physical sphere, *e.g.*, vomiting, nervous cough. Relief can be afforded by means of speech, to such states arising in this way. This has been long recognised in the Roman Church, by the institution of confession. Benefit may, however, be obtained from confession without a priest or absolution. To this whole group of cases, the authors give the name of "*Rétentions Hysterie.*" There is another group of cases which Freud has described under the title of "*Abwehr-Neuro-Psychoses.*" By "*Abwehr*" is meant the voluntary suppression of painful concepts, of concepts which a man feels to be injurious to his life's activities, or to his self-esteem. It is not easy to understand how a concept may thus be voluntarily forced out of consciousness, and cut off from its normal associations, but the corresponding positive process is sufficiently established, *i.e.*, the concentration of the attention on a concept, and we know just as little how this is actually accomplished. Concepts separated from consciousness remain also cut off from the wear and tear of cerebral activity, and preserve their emotional accompaniments unaltered.

A third group of conscious states remains withdrawn from the wear and tear of thought, not voluntarily, but because they are forgotten, having originated in, and having been associated with emotions in states like hypnosis and the conditions allied to it, for which amnesia exists in the waking state. These are of great importance in the study of hysteria.

Conditions resembling hypnosis occur in the so-called hysterical hypnoid states. The authors describe the occurrence of states like hypnosis in hysteria, whose importance consists mainly in the amnesia present in them, and in their power to produce later that division or splitting up of the normal consciousness which is so important in severe hysteria. Yet they limit the importance of these states to the origination of somatic phenomena from idea, the transformation of psychical into physical innervation. Division of consciousness may also occur through voluntary amnesia in "*Abwehr-Hysterie*," by which concepts, or group of concepts, are shut off from the interchange of normal association.

These hypnoid states in hysteria alternate with waking states, and vary in frequency and duration; the dreamy condition to which they give rise often deserves the name of hysterical delirium. They differ from induced hypnosis only in their spontaneous origin and, as the name given implies, in being less well defined.

The mental contents of hypnoid states are shut off from those of the normal or waking life by amnesia, so that they cannot undergo correction, and, owing to loss of criticism and control by other percepts or concepts, may give rise to the most deranged and persistent hallucinations. A complicated irrational or "symbolical" nexus may arise between the pathological phenomena and their exciting cause, often dependent on ludicrous sound-similarities and word-associations. Hysterical symptoms may be restricted to the hypnoid condition, so long as this alternates with the waking state, and be intensified by repetition. The mental image, or idea, which is the cause, of the condition, never emerges clearly into consciousness. Change of psychical into somatic symptoms may take place during these hypnoid states.

In one case the origin of these states arose out of fits of reverie, which were the result of long-protracted anxiety. The author thinks they often arise in brooding over love disappointments, and during long hours of nursing sick relatives. It is possible that such states may arise spontaneously in those hysterically disposed without the co-operation of the emotions. It might be thought that an intelligent patient, capable of self-observation, would recognise the connection between idea and hysterical phenomena. The inner causal nexus would be obscure, but the outward connection would be recognised. Patients, however, as a rule, deem themselves to suffer from independent corporeal phenomena. The authors believe that their observations show that the *psychical trauma* remains in memory for a long time as an active working (irritative) agent, outside the patient's consciousness, and comparable to a foreign body. A division of consciousness or of the psychical activity results, and is of fundamental importance in the elucidation of complicated hysterical phenomena.

Sub-conscious Perceptions — Division of Consciousness.—The existence of sub-conscious mental states, or perceptions is undoubted. Understanding by "sub-conscious" states those in the background of consciousness, they make up a large part of the psychical accompaniments of normal brain action. The ability of perceptions to affect consciousness is largely dependent on the extent to which they are associated with emotion. If they at once set free a motor act, by passing at once into motor channels they lose their clearness and intensity as perceptions. The following example is given of the mode in which such perceptions may give rise to hysterical symptoms. A man whilst eating sustains some intense emotion to which there is no corresponding

reaction (*e.g.*, by speech, action or weeping, &c.) Subsequently each attempt to eat excites loathing and vomiting, and relief is not obtained from these symptoms until an outlet, by speech or some other means, has been afforded to the pent-up emotion. The patient is ignorant of this connection between cause and symptoms, which may only be discovered with difficulty by aid of hypnotism.

In such individuals presentative or representative states may be conscious or sub-conscious; they may or may not be capable of affecting clearly defined consciousness; thus is constituted a division of the psyche, which, in hysteria, is divided into two relatively independent parts. The authors do not say that all hysterical phenomena arise in this way, but rather that "that division of the psychical activity, which is so striking a feature in the known cases of double consciousness, exists in rudimentary fashion in every marked case of hysteria, and that the tendency for this dissociation to occur is the fundamental phenomenon of the neurosis." Professor Janet regards this division of psychical activity (personality) as dependent on innate psychical feebleness. In the normal man it is only when the attention is concentrated on one subject, or when the image or idea is exceptionally powerful, that it occupies the whole field of psychical activity, otherwise it arouses, by association, a number of others, which support or inhibit it. In hysteria, however, each one of any degree engrosses the whole psychical activity, and is thus shut off from associative interchange. The author does not agree with this view, that there is innate feebleness of mind, which he thinks is negatived by fact that the most highly gifted persons may be hysterical, but considers that the due action of the mental powers may be interfered with, or enfeebled, by the co-existence of two heterogeneous groups of perceptions or ideas, causing loss of power by diversion.

The co-existence of day dreaming, or of frequent strong emotional states, with normal psychical activity, tends to a pathological division of the psyche. If the two co-existing groups of conceptions no longer have a similar content, if one group is sub-conscious the division is accomplished; hence arise hypnoid states.

How far does the lately-acquired knowledge of division of consciousness aid in the understanding of hysteria? First, many apparently physical phenomena can be traced to psychical causes of which the patient is not fully aware. The sub-conscious representations, though never, or very rarely, entering into the waking thoughts, yet influence them, and also influence the processes of

association. Thus they determine the disposition, and render comprehensible the "tempers," and motiveless and inexplicable "humours" of hysterical patients. The emotions are reinforced by the sub-conscious representations, and, from the oscillation between primary and secondary states, the psychical powers are weakened, and the patients seem devoid of energy, childish, or even imbecile. In hemianæsthesia the loss of impressions from one half of the body tends in the same direction. Further, the increased susceptibility to suggestion is explained by the poverty and incompleteness of the sub-conscious separated part of the psyche, and the disturbance of association.

With regard to the original disposition in, or mode of development of hysteria, the author, as above stated, rejects Janet's view of innate psychical weakness, and substitutes an abnormal irritability of the Central Nervous System, through which the sensory, vasomotor, and visceral apparatus are abnormally accessible. Thus arises hyperalgesia of a part to which attention has been directed by some slight lesion; *e.g.*, hysterical joints, palpitation, nervous asthma, &c. The psychical origin of hysterical anæsthesias, analgesias, and contraction of visual fields is, he thinks, not yet established. The stigmata, the especial hysterical symptoms which form the diagnostic basis of hysteria, arise out of the fundamental disposition, and do not originate in idea. Next to them come those phenomena which are "ideogenous," and are simply the conversion of emotional excitations into somatic (physical) symptoms. The author lays stress on the fact that the most frequent and most powerful emotions to suffer this "conversion" are the sexual ones; that the sexual factor is by far the most potent and most pathologically fruitful in the production of hysterical phenomena. The observation of the ancients expressed in the word hysteria is nearer to the truth than the new views which put sexual disturbance almost in the last line. Next to the sexual factor the most important is terror—the essentially traumatic hysteria. The hypnoid states, or tendency to autohypnosis, form the third constituent of the hysterical disposition. The origin of these states is doubtful, and how sexual influences are concerned is not known. The mode of origin of division of consciousness in hypnoid states has been alluded to, and these states are responsible for the origin of hallucinations and of motor acts independent of the conscious will.

Psychotherapy of Hysteria.—The method of treatment followed has been described in BRAIN (*loc. cit.*); briefly it consists in getting the hypnotised patient to fully express in words the mode

of origin of his symptoms with the accompanying circumstances of the onset. Dr. Freud names it the *cathartic* method of treatment, and remarks that two difficulties had to be met at the outset: first, that all patients could not be hypnotised; and second, that a diagnosis could not be based on the results of treatment. For all hysterical disorders are not curable by this means, and some other neuroses are. He, therefore, resolved to investigate and treat all neurotic patients in the same way, and to allow the diagnosis of hysteria to depend on the result of the enquiry. As a result of this investigation he was forced to the conviction of the paramount importance of sexual disorders in the ætiology of the neuroses; and further than this, that different forms of sexual disorder produce different forms of neurosis. He differentiates the following forms of neurotic disorder:—

(1) Neurasthenia, presenting constant clinical features, in which a psychical mechanism plays no part.

(2) "Zwangsneurosis," characterised by uncontrollable and frequently-returning thoughts and ideas, and having a similar etiology to hysteria. Imperative ideas.

(3) "Angstneurosis," split off from neurasthenia, and characterised by anxious forebodings, morbid fears, and hyperæsthesia to pain. It is not of psychical origin, but influences psychical activity, arising through repeated physical overstrain, itself, again, of sexual origin. It is to be distinguished from hypochondriasis, in which there is a constant dread of bodily illness.

(4) Hysteria.

The above neuroses exist frequently in combination in the same person. Neurasthenia and the "Angstneurosis" are most frequent in the pure form. It is scarcely possible to consider hysteria apart from sexual neuroses. He thinks hysteria may in every respect be treated of independently from other neuroses, except from the point of view of therapeutics. From his own point of view it is most important to separate clearly the part played by hysteria in the formation of a mixed neurosis, for the cathartic method of treatment is effectual in hysteria, is of no avail in neurasthenia, and only to a limited extent in the "Angstneurosis." He does not claim to remove all hysterical symptoms, but attributes failures to difficulties in dealing with individual cases rather than to faults in the principle of the method. The cathartic method treats symptoms and not causes. It is most effectual in dealing with symptoms remaining after an acute hysterical paroxysm; here it meets all indications, and gives

prompt and enduring results. In acute hysteria, *i.e.*, period of active production of hysterical phenomena, it is of less value; because the symptoms, if removed, soon recur. In monosymptomatic hysteria and in chronic cases, with continuous production of hysterical symptoms, the cathartic method is especially valuable. Dr. Freud further insists on the necessity for the fullest confidence between doctor and patient, if success is to be attained; that in his hands hypnosis has led to no bad results; and that his method of treatment may often be combined with the Weir-Mitchell treatment, with most advantageous results, which could never be obtained from the latter alone. The author, at the outset, was confronted with the difficulty that all patients are not hypnotisable. He found that in many of these cases, by repeated close questioning, some information could be obtained of the primary origin of the symptoms. To get information in this way required great mental exertion, in order, as he supposes, to overcome a psychical resistance in the patients, tending to prevent the emergence of these pathogenic ideas into consciousness. He notes that all these pathogenic ideas have the common attribute of painfulness; they were attended with emotions of shame, remorse, or injury. In the evolution of the neurosis this painful impression had been "converted," forced out of consciousness, but had left traces of its presence. The same power which had originally forced out of the conscious life the painful impression now resisted his efforts to revive it in memory. Hence, in hysteria, the not knowing implies disinclination to know; and the object of treatment should lie in overcoming the resistance to normal processes of association. As a result of these investigations, the author was led to adopt the following method of procedure, to which he attaches no specific importance other than convenience. The patient lies down with his eyes closed, and the observer, exercising light pressure with his hand upon the patient's forehead, tells him that during this pressure a picture (visual memory) will rise before his eyes, or a remembrance of some past event will be recalled, which he must describe at once without reserve, and as fully as possible, of whatever nature it may be, even if it appears to him to be disagreeable, trivial, or not to the purpose. In this way a knowledge of the pathogenic concept is obtained, occasionally directly, but more often after a long chain of associations has been started, which finally leads to the desired result.

Perseverance is necessary to success, the method may fail the

first time and afterwards be completely successful. It is essential that the patients should simply report what they visualize, or otherwise recall, and not their own reflections. Visual memories are most easy to deal with. The vision fades from memory, after its description by the patient, unless there still remains something of importance to be elicited in connection with it, in which case it stays obstinately before him.

As a rule the method is a trustworthy one, and succeeds in every case; it must be combined with close observation of the patient's expression; one soon learns to distinguish the expression of mental rest, which follows on a successful employment of the method, from the state of strain and emotion existing previously. The full results to be gained by the procedure are only reaped at the cost of much time and patience. After a time the contrast between the results at the first and those attained at later séances are very striking. The patient can be intellectually interested in the process, and thereby further advance made. If the question were asked whether recourse to hypnotism would not render the carrying out of the "cathartic" method more thorough and easy, the author, so far as his experience goes, would reply in the negative.

In a former paper the authors made the classification of hysteria into

1. "*Abwehr Hysterie*"—acquired hysteria, in which an unbearably painful concept is forcibly expelled out of consciousness and transformed into some somatic disturbance.

2. "*Hypnoid Hysterie*," in which there is a division of consciousness, one group of concepts being completely separated, or shut off, from the ordinary consciousness.

3. "*Retentions Hysterie*." The authors are not now prepared to recognise the second form as distinct in origin from the first, and do not finally pronounce upon the third form.

They go very fully into the method of carrying out the above mode of treatment, and the difficulties likely to be met with; the analysis of the symptoms, and of the psychical changes on which they depend, proceeding step by step until a full acquaintance with the mental state of the patient, and of the conditions which originally gave rise to the neurosis and of the subsequent stages in its development, has been gained.

I have in the above endeavoured to give as concisely as possible the authors' views. The most important points in them seem to be that they afford an explanation for the origin of some of the somatic symptoms of hysteria in psychical causes. They still

leave unanswered, however, the mode of origin of the fixed "stigmata," such as hemianæsthesia, contraction of visual fields, &c. Perhaps the strongest arguments in their favour are, first, the approximation afforded on their views of ordinary cases of hysteria to cases of traumatic hysteria, in which the cause, the way in which it acts in producing hysterical symptoms, and the nature of the symptoms so produced, can be most easily followed out and studied; and, secondly, that due importance is attached to the undoubtedly great part played by the emotions in the evolution of hysterical symptoms, and some explanation given of the way in which they do this. It may further be added that their theories give, in psychological terms, an explanation of some of the characteristics of hysteria, long established by medical observation, and some of them matters of common knowledge. It is interesting to note a return, in part at least, to the old theory of the origin of hysteria in sexual disorders, especially as the tendency of late years has been to attach very much less importance to them. The authors adopt the theory of a division of consciousness as accounting for many hysterical symptoms and states; in the ordinary run of hysterical cases, this, if present, must be so in a very rudimentary form. They also pre-suppose, as underlying hysterical phenomena, an irritable state of the nervous system. It is in what exactly constitutes this "irritable state," and how it arises, or, if we prefer so to call it, the "innate mental feebleness" of Janet, that the key to the problem of hysteria is to be found; and it is just as to this point that our knowledge is most severely limited.

The book is written in an interesting style, and bears the stamp of originality, and of much patient and painstaking observation. Into the question of the advisability of penetrating so intimately into the most private thoughts and concerns of a patient I do not enter; it would seem likely that the patients would, in many cases at least, strongly resent it. The necessity of bearing in mind, in studying hysterical patients, the great readiness with which they respond to suggestions, may be reiterated, as the weak point in the method of investigation may perhaps be found here. The danger being that in such confessions the patients would be liable to make statements in accordance with the slightest suggestion given to them, it might be quite unconsciously given to them, by the investigator.

J. Michell Clarke.

Traité clinique et Thérapeutique de l'Hystérie d'apres l'Enseignement de la Saltpêtrière. Par le Dr. GILLES DE LA TOURETTE, Professeur Agrégé à la Faculté de Médicine de Paris, Médecin des Hopitaux, &c. Preface de M. le Pr. J.-M. CHARCOT. Seconde Partie. Hystérie Paroxystique avec 63 figures dans le texte et un portrait à l'eau-forte du Professeur Charcot. Vols. I. and II. pp. 1146. Paris: E. Plon, Nourrit et Cie, 1895.

THESE two volumes form the second and third of M. Gilles de la Tourette's work, and bring it to a close. They deal with the paroxysmal manifestations of hysteria, defined as the episodes or incidents occurring on the general basis afforded by the presence of hysterical stigmata, and characterised by suddenness of onset and of disappearance. The attack may be acute or prolonged, the latter forming the *état de mal*, or, in the sphere of mind, may constitute a permanent "second state." It may be said at once that the book is a very complete one, and that every kind and degree of hysterical manifestation is here treated at length. In this notice the author's views, which embody those held at the altpêtrière, will be given on those features of hysteria which are of most interest and importance, or about which there is considerable divergence of opinion. The first volume, of which a digest will be found in BRAIN, 1892, pp. 522 *et seq.*, dealt with "normal" hysteria, *i.e.*, the ordinary state of hysterical persons between the paroxysms; the so-called "stigmata," sensory and motor, being taken as the evidence of the presence of the hysterical state.

In treating of hysterical fits Charcot's description of the paroxysm in hysteria major is adhered to, and his well-known table of phenomena, and P. Richer's beautiful drawings, are reproduced. At the same time the author admits that the fully-developed typical attack as thus described is rare; he also admits the part which suggestion may play on other hysterical patients in the same ward, with a sufferer from these fits, but thinks that the description should be retained to aid in the elucidation of the incomplete fits. He combats the view that has been brought forward by Bernheim and others, that these fits are a growth of the Saltpêtrière, and peculiar to it, by quoting published cases of other authors, notably Andrée and Knoblauch's account of the occurrence in German soldiers of fits corresponding exactly to

Charcot's description. It must be admitted, we think, that attacks in the complete form are very rarely observed, and are a matter rather of theoretical, than practical importance. The author gives a long and vivid description of a patient in one of these severe attacks. A short account of the ordinary, milder, hysterical fit, such as is commonly met with, is desirable. Whether it is true that the milder attacks represent incompletely the more severe ones, some phases of the latter only being present, must be difficult to prove satisfactorily. Certainly in the immense majority of hysterical patients only the minor attacks occur, and only very distantly resemble the severest form. The greatest point of resemblance lies in the frequent occurrence of a state resembling the somnambulistic or hypnotic condition—the third period in Charcot's scheme—in which the patients react readily to suggestion from without.

Amongst minor points the author says that convulsive attacks, though less frequent in men, when present are severe, and that the period of delirium is the most marked feature of the fits in children, and may be the only one. He divides the aura into (1) prodromal phenomena, and (2) aura proper. The most interesting and varied of the prodromal phenomena are in the intellectual or psychical sphere, and have generally a sombre character. He gives elaborate researches, which involved much labour and were carried out with due precautions to avoid error, on the nutritive changes during a convulsive attack, as shown by the urine. All the urinary excreta are diminished relatively to the normal during an hysterical, whilst they are augmented during an epileptic fit. The urine examined for twenty-four hours from the onset of an hysterical fit shows a diminution in the amount of urea and total solids, whilst the ratio of the earthy to the alkaline phosphates is changed, the earthy being passed in increased ratio; the normal proportion being 1 to 3, in hysteria the amounts passed of each may be equal. In the cases investigated, variations in amount of food were carefully excluded. The whole formula should, however, be taken into consideration, and not merely the inversion of the ratio of the phosphates. He replies, at considerable length, to those who have combated the accuracy of this statement; and the conclusions arrived at appear to be justified by the data given. Considerable stress is laid on this as a method of diagnosis between hysteria and epilepsy.

Do hysterical fits ever terminate fatally? This subject is discussed in an interesting section, in which the historical cases

of death from hysterical seizures are reviewed. It comes out that in nearly all the cases the accuracy of the diagnosis is open to question; and the author concludes that a fatal termination in the course of an hysterical fit, whether simple or prolonged, is of the rarest occurrence; and that when it does occur, attacks of laryngeal spasm most frequently cause it. Finally, for a fatal result to take place, the co-existence of organic disease seems to be necessary, the fit being merely an adjuvant cause. Hysterical fits may be very rapidly repeated at short intervals, during which the patient suffers from the effects of the paroxysms (*état de mal*). One characteristic of these attacks is the rapid loss of weight that occurs, the loss being made up almost as rapidly when the attack is over. The urine shows the characters given above, and the quantity passed is of some prognostic importance, as at the onset there is a fall in the amount, which remains stationary during the attack at a lower level than health, and towards its termination begins to rise again to the normal. This general rule is, however, vitiated by exceptions, due to the frequency of relapses. The chapter on lethargy, or hysterical sleep, or trance, is particularly valuable. It begins with an account of the history of the subject; for in all ages this peculiar condition has excited great interest, and in many of the most ancient cases the diagnosis is quite clear. With regard to diagnosis, the doctor may be called to see a patient apparently comatose, but really in hysterical sleep. In women a history of hysterical attacks can generally be obtained, and other hysterical symptoms observed; but in men such a condition may be the first manifestation of the neurosis. The onset may be sudden or gradual, and is generally preceded by prodromal symptoms. Frequently active hysterogenic zones are to be found, together with general anæsthesia. Even if there is general muscular relaxation, trismus, or rapid intermittent contractions of the eyelids are present. The author recognises attacks which last only some seconds or minutes! They may certainly last for weeks, but observations of a duration of months or years he considers doubtful. The patients are not necessarily fully unconscious; instances are on record where they have heard and appreciated the preparations for their burial without being able to give any sign. The attack terminates suddenly by a convulsion or an outbreak of tears or laughter. Death never seems to occur; the one case on record being of doubtful nature. Similar changes in the urine to those above described as occurring during the fits are present, and are independent of the amount of food taken.

Under the name of katatonia, Kahlbaum described what he believed to be a special disease. In this a stage of melancholic depression precedes a stage of excitement, which is followed by a catatonic stage, presenting the characters of melancholia with stupor. In the latter motor phenomena are present, which bear a close resemblance to hysterical sleep, with cataleptiform contractions. The patient remains in a lying posture, with abolition of general sensibility and of the special senses, vibratory tremor of the eyelids, trismus and cataleptiform contractions of the limbs. This condition (catatonia) seems further to be sometimes associated with hysteria. The former, however, is distinguished by a history of antecedent mental trouble, whilst the onset of hysterical sleep is generally abrupt, and, unlike catatonia, never ends in dementia or death. M. Gilles de la Tourette does not admit that there is any well authenticated case in which hysterical sleep has lasted more than from five to six weeks consecutively, whereas the catatonic condition has been known to last for eleven years.

Catalepsy itself he does not consider, so far as its objective phenomena are concerned, to belong to hysteria; it is to be regarded as a group of symptoms, which may appear under various conditions, *e.g.*, in hysteria, in which case it frequently accompanies the fits, or in mania and melancholia. When it succeeds hysterical fits the different cataleptic attitudes are assumed under the influence of hallucinations. In hysteria catalepsy may be merely mixed up with other phenomena of the attack, or may be the dominant and apparently primary symptom. The former is by far the most frequent. Prolonged cataleptic conditions in hysteria are to be regarded as attacks of prolonged hysteria. Here, again, both in diagnosis and prognosis the author largely relies on the characters of the urine.

With regard to hypnotism, the author urges the view held at the Saltpêtrière, that hysterical persons only are really hypnotisable. General experience has, however, shown that the phenomena of hypnotism are not confined to the hysterical. Somnambulism he regards as merely a manifestation of hysteria, and regards the "second states" of double consciousness as a prolonged hysterical somnambulism, in which the convulsive phenomena play only a minor part.

In this connection the curious affection, called "ambulatory automatism," is mentioned. Cases are met with in which the patients wander off on some vague but uncontrollable impulse,

leaving their homes and avocations, and after a longer or shorter time, return to their normal state of mind, with no memory of anything that has occurred in the interval. If the attacks occur often they lead a kind of dual existence. This condition has long been known to occur, in greater or less degree, after epileptic fits. Charcot and Pitres first described it in hysteria. According to them, the hysterical differs from the epileptic variety, chiefly in not being amenable to bromides, and in the fact that, if the patient is hypnotised, in the hypnotic state he remembers all that passed during the period of "automatism." Unfortunately, no investigation of a patient whilst actually in this condition has yet been made. There is no doubt of the affinity of these states to somnambulism; and the author further regards them as equivalent to the third and fourth period of the hysterical fit. The view which regards hysterical somnambulism and second states, as modified periods of the fits, seems to rest chiefly on simularity, and is, perhaps, a point which requires further proof.

The views as to the nature of hysteria, held by M. Janet and his fellow-workers (BRAIN, vol. xvii., p. 129) do not commend themselves to the author, and he dismisses them with a brief reference. He thinks that they do not account for the physical phenomena of hysteria, and that in place of accurate clinical observation, these writers have merely applied to hysteria preconceived psychological theories. Some of their experiments have, however, thrown remarkable light on the true nature of hysterical anæsthesia, &c. There is a very full account of cutaneous trophic disturbances; the author states he has been careful to accept only facts of undoubted authenticity. Vasomotor disturbances are so common as to form a special feature of the neurosis. A perusal of the cases of the various skin eruptions met with in hysteria, suggests that great care and discrimination is needed before directly attributing them to the neurosis.

The occurrence of spontaneous ecchymoses, or hæmorrhages, from the skin and viscera, is so interesting that I give a little longer notice of M. G. de la Tourette's account of this subject. One of the most remarkable and best authenticated cases of hæmorrhages from the skin is that of Louise Lateau—*la stigmatisée Belge*—studied by Bourneville. In her case the bleeding points corresponded to the stigmata of crucifixion, and bore a remarkable resemblance to the celebrated bleeding stigmata of St. Francis of Assisi. This rare phenomenon is not confined to hysteria, but has been most frequently observed in it; at least one case in man is on record; and, in women, although it is often related

to the suppression or appearance of the menstrual periods, it has no constant connection with them. In many patients the bleeding seems to be due to the secretion of a blood-stained perspiration, rather than an actual hæmorrhage; but in others the appearance was that of ordinary blood. The bleeding has most often taken place from an erythematous or vesicular patch, and sometimes from an indolent ulcer. In one case the hair follicles were the seat of hæmorrhage, which was confined to the hairy parts of the body, little drops of blood forming around the roots of the hairs; and cases are recorded when the bleeding took place from the ceruminous glands of the external auditory meatus. The cutaneous hæmorrhage is often accompanied by epistaxis, hæmatemesis, or melæna. Although the quantity of blood lost has been considerable in a few cases, no danger to life seems to have ever occurred. Pains, or sensations of burning, or an hysterical fit, sometimes precede the hæmorrhage. The reader will find ample information on this subject in the book. Hypnotism has, perhaps, thrown some light on this obscure subject, by showing that cutaneous lesions may be the result of suggestion. For instance, a postage stamp was stuck on a patient's shoulder in a spot that he could not reach; he was hypnotised, and told that the stamp was a blister. He was placed under close surveillance during the rest of the day, and sent to sleep for the night; in the morning a blister was found. Again, blisters were placed on the chest of a tuberculous patient, and on both arms of a hysterical patient; the latter was told during hypnosis that the plaster on the right arm would not raise a blister. Care was taken to watch this patient closely; and it was found that blisters duly appeared on the first patient's chest, and on the second patient's left arm, but not on the right. An *hæmoptysis* due to hysteria is now described. It occurs in men as well as in women, which negatives the view that these hæmorrhages are vicarious of menstruation. The author thinks its more frequent occurrence at the menstrual periods due to the fact that convulsive attacks, which provoke hæmorrhage and other hysterical vasomotor phenomena, are so common at these times. Hysterical hæmoptysis may occur during the fits, or in the intervals. Such patients often suffer from painful points or areas in the chest wall, and from an hysterical cough. In most patients the physical signs are much less than would be expected from the abundance of the hæmoptysis, intensity of cough, and aspect of the patient. Local signs, such as crepitation, may come and go from day to

day. The author thinks that pulmonary congestion of hysterical origin exists, characterised by appreciable signs on percussion and auscultation. As a rule, which is not invariable, fever is absent; and in most cases the hæmorrhage is stated to be more abundant than in pulmonary tuberculosis. The author points out the great difficulties in diagnosis from the latter. He notes the frequency of phthisis in the hysterical; and thinks that there is no proof of the view that has been urged by some writers that hysteria is truly antagonistic to, prevents the development, and retards the course of phthisis.

Hysterical hæmatemesis has been observed under the following conditions:—An attack of acute pain and swelling in the epigastrium, the pain increasing and radiating into the back, comes on with the ordinary symptoms of the hysterical aura, *i.e.*, palpitation, præcordial anxiety, sensation of constriction of the œsophagus, giddiness, noises in the ears, and beating at the temples; this is followed by vomiting of blood, once or twice repeated; the patient passes then into a hysterical, lethargic state, which is not due to syncope from loss of blood, because it occurs when the loss of blood has been very slight. The blood may be pure or mixed with fluid from the profuse salivation, which often accompanies the attack. If these hæmorrhages are large or frequently repeated, as they sometimes are, for five or six days, the patient falls into a profoundly anæmic state. In the course of some investigations into the diagnosis between hysterical hæmatemesis and that due to simple round ulcer of stomach, the author found that of eighteen patients (thirteen women, five men) suffering from well-marked symptoms of gastric ulcer, six were hysterical. He suggests that hysteria may be a determining factor in the etiology of round ulcer; and, reasoning from the analogy of the above-described trophic cutaneous lesions in hysteria, supposes that an ecchymosis (hysterical) of the mucous membrane may, under the action of the gastric juice, be converted into an ulcer.

Hitherto it has been generally held that, in a doubtful case of severe gastric pain, the occurrence of well marked hæmatemesis would decide in favour of a diagnosis of ulcer. The hyperæsthesia over the epigastrium, and excessive epigastric reflex, so commonly present in gastric ulcer, can hardly be adduced as evidence of hysteria. Hysterical girls are very often anæmic; gastric ulcer occurs most frequently in the anæmic; and one would expect, therefore, that gastric ulcer should be sometimes associated with hysteria. I think that clinical experience in this country, at

least, does not point to any closer relationship of the two affections; cases of gastric ulcer do not, as a rule, present evidence of hysteria. In the case of both hæmatemesis and hæmoptysis, an hysterical origin for it could not be accepted as satisfactory without the positive proof furnished by *post-mortem* examination; seeing that both gastric ulcer and pulmonary tuberculosis may be present without being recognised clinically. Healed tubercular lesions are found in the lungs *post-mortem*, when nothing in the patient's history pointed to a previous tubercular affection; and ulcers, healed or not, are also occasionally found in the stomach, whose existence had been unsuspected during life. In view of these facts it is hardly necessary to add that a diagnosis of hysterical hæmoptysis, or hæmatemesis, must be an uncertain one; and, if made, could only be acted upon with the greatest caution.

There seems no doubt that muscular atrophy may occur in hysteria. Most cases have been in men. Generally it is a complication of paralysis or contracture, and other trophic disorders are often present. The onset is fairly rapid; and it most commonly affects the whole limb, but sometimes only a part of it, such as the small muscles of the hand. The atrophy is accompanied by fibrillary tremor. The electrical change is a diminished reaction to both forms of current. Aid in diagnosis is given by the atrophy following on paralysis or contracture, with disturbances of sensation and other hysterical manifestations. There is a good account of hysterical fever, to which the reader may be referred, as this subject was treated fully in BRAIN, 1894, p. 196.

The section of hysterical paralyses and contractures is very full and complete, and it opens with an interesting historical introduction. Full justice is done to the work of Todd and other English writers on the subject. With regard to hysterical hemiplegia, the old diagnostic point as to absence of affection of the face, though generally, seems not to be universally true, many observations being now on record of paralysis of the lower part of the face. It is, however, rare and as a rule, slight, it varies from day to day, and individual movements may be dissociated and paralysed. It is often accompanied by spasm of the muscles of the opposite side of the face.

Labio-glossal spasm is more common than facial paralysis in hysteria; in the great majority of cases it accompanies hemiplegia, generally on the same side, but sometimes on the opposite. The affected muscles are affected with a rapid clonic tremor. The spasm has a tendency to spread to the neck muscles, especi-

ally to the platysma myoides. Trismus, common in the hysterical fit, is rare as a persistent affection, and is then generally accompanied by spasmodic contraction of other muscles. This labio-glossal spasm is little marked during repose, but comes out on movement. There is slight derangement of speech and deglutition. The excessive deviation of the tongue is a diagnostic feature; when the patient is told to protrude the tongue, it is bent round like a hook, so that the tip bulges out the cheek. Aid in diagnosis is also given by the presence of anæsthesia over those parts of the face and tongue affected by muscular spasm. Like spasm, hysterical facial paralysis is most obvious on movement, and very little marked during rest. There is no change in the electrical reactions in either spasm or paralysis. Both are generally transitory, but one case lasted three years, and showed no tendency to cure.

Hysterical hemiplegia, monoplegia, and paraplegia are fully discussed. With regard to monoplegia, it is said that the prognosis is variable, but bad, in male adults. Monoplegias, generally, are apt to persist (see BRAIN, vol. xvii., p. 294), and to be attended with trophic troubles. The extent of accompanying anæsthesia varies; so long as any anæsthesia is present, the paralysis is apt to return. In two cases of paraplegia of spastic type, in which the diagnosis of hysteria was not doubtful, incontinence of urine was present. Two cases of bed-sores occurring in the same affection are mentioned. In the differential diagnosis of hysterical from organic hemiplegia, stress is laid on the frequency of sensory disorders in the former; on the general absence of affection of the face, whilst if the facial muscles are affected, spasm, and not paralysis, is generally present; and on the fact that the tendon-reflexes are normal.

The author believes in a mode of action of hysteria, which he terms "auto-suggestions organiques." An example will best explain what is meant by this term. MM. Brissaud and Lamy report a case of brachial monoplegia, of historical origin, in a patient suffering from lead-poisoning; the supinator longus being unaffected, just as in ordinary lead palsy. They quote a similar case described by M. Potain, and conclude: "Hysteria has smitten the parts affected by lead poisoning." Similarly, the author says, that he has often observed hysterical hemiplegia and brachial monoplegia in syphilitic patients, who had been more or less affected by cerebral syphilis. Instead of the hemiplegia or monoplegia of organic origin, which had been cured by appropriate treatment, hysterical hemi- or monoplegia had

appeared, for whose provocation and localisation the organic lesion was responsible. The frequency with which hysteria complicates organic nervous disease is now generally accepted; and, *a priori*, one might expect that those parts previously paralysed from organic lesion would be more likely than others to be affected in a subsequent outbreak of hysteria. But that the hysterical should occupy the exact field of the organic paralysis to the extent described above seems to require further proof. In a case like the one of lead-poisoning, such a distribution of the paralysis would make the diagnosis of its hysterical nature one of extreme difficulty and of some doubtfulness.

There are good descriptions of hysterical paraplegia, pseudo-tabes, and of the various forms of contraction affecting the upper and lower limbs, especially of the hand and foot; this section on contractions having a number of excellent illustrations. The author brings forwards evidence and quotes authorities in favour of the view that many cases of tetany, and especially those cases of so-called essential tetany, in which no cause can be traced, are of hysterical nature. He thinks that a large part of the essential contractions of the extremities or tetany, in both mild and severe forms, are due to hysteria, and that hysterical tetany may show itself in epidemic form in children.

Following Charcot, three forms of astasia-abasia are distinguished. In this affection movements of standing and walking are alone affected; rarely is there any affection of sensation or of the muscular sense; and the reflexes and muscular nutrition are unimpaired. (1) Paralytic or paretic, in which there is weakness or difficulty in walking, or the gait is stiff and constrained. (2) Choreiform (type of flexion, Charcot), in which there is sudden bending or giving way of the legs, with consequent violent compensatory muscular contractions to prevent falling. (3) "Trépidante," in which walking is rendered difficult by contradictory movements, which stiffen the lower limbs and produce a sort of tapping, stamping, or clonic spasm. This latter, in exaggerated form, gives the saltatory variety long described as saltatory spasm. The paralytic form may give rise to difficulty in diagnosis from cerebellar tumour, especially as there may be marked vertigo. The pathogenesis of the affection is referred to a disorder of the complex co-ordinated spinal mechanisms for standing and walking.

The author gives a learned and exhaustive chapter on the various forms of cough, and of simulation of cries of animals in

hysteria. Some of the accounts of epidemics of this nature in former times are very curious. In one instance the nuns in a convent took to miauling like cats; the noise speedily ceased when the magistrate stationed a company of soldiers at the door, with orders to enter and chastise the miaulers!

Yawning, sneezing, and laughing are known to occur in paroxysms in the hysterical. One patient is said to have yawned eight times a minute, or 7,200 times in fifteen hours. These paroxysms may end in true convulsive seizures.

In hysterical laryngeal affections it is often very difficult to say whether we have to deal with a paralysis of the abductors, or spasm of the adductors; as in the case of the ocular and facial muscles, paralysis of one set is apt to be accompanied by spasm of their antagonists. A form of dyspnœa is met with in the hysterical, which, if its existence is not known, may lead to errors in diagnosis. The name, tachypnœa, proposed by Charcot, is more suitable than dyspnœa. The respirations may be 170-180 to the minute; they are shallow, and not noisy; the auscultatory signs are normal; there is no cyanosis, and the pulse is not quickened; the patient betrays no anxiety. The attacks last from three to four hours; are almost always preceded by symptoms like those of the aura of an hysterical fit, and often end in an outburst of tears.

In hysterical gastric affections, vomiting is considered to be the predominant symptom, and attributed to hyperæsthesia and spasmodic contraction of the stomach. The vomiting is constant, the food being rejected without effort or pain as soon as swallowed. All the food is not vomited, but the amount retained is not sufficient to prevent emaciation. The event is favourable, but there is a great tendency to relapse. The author attributes both hysterical vomiting and gastralgia to the presence of hysterical stigmata in the digestive tract. In gastralgia he supposes that there is an hyperæsthesia of the gastric mucous membrane; and from this point of view he recognises two clinical varieties: (1) cases in which the stomach is the seat of an hyperæsthetic and hysterogenic zone, and the skin over it is also the seat of anæsthesia or hyperæsthesia; the above zones are only rendered active as the sequel of a convulsive attack, or of irritation of an ovarian zone. (2) Cases less common, in which the hyperæsthetic gastric zone acts primarily. Food then excites an attack; and if not at once expelled, the symptoms increase in severity until they may even end in actual convulsions; pain being, however, the most striking symptom throughout. After such an attack is over, the

stomach becomes tolerant, and food is retained and digested. Marked changes in the composition of the gastric juice are not found.

In hysterical anorexia the psychical element predominates, though anæsthesia of the stomach may aid in producing the symptoms. The two psychical factors are the loss of sensation of hunger and of all wish or desire to eat. In the earlier stages of the affection, the patients display an unusual and morbid activity; later, they fall into a semi-comatose state, in which the only will-power shown is the refusal to take food. The fact that the stomach may one day not tolerate a drop of water, and on the next be able to manage a full meal, shows that it is not primarily affected, and that the disorder is a mental one. Relapses seem not to occur; a point in which this affection differs from similar conditions in the insane, and also from other hysterical disorders. The urinary excreta, liquid and solid, vary according to the amount of food assimilated. Death very occasionally occurs from pure inanition, sometimes from the supervention of tuberculosis. Sometimes the patient has regained the desire to eat and to live, but too late, the power to assimilate nourishment being lost. The author thinks it necessary, in view of the statements that have been made that hysterical anorexia is not attended with wasting, and is never fatal, to give some tables of cases, showing the daily loss of weight, and to emphasise the fact that the disease may undoubtedly, though rarely, have a fatal termination. Speaking generally, digestion is retarded, and the amount of HCl in the gastric juice deficient, but there are not at present sufficient data to state exactly the alterations in the gastric juice.

The most important affections of the intestines in hysteria are hysterical tympanites, meteorism, or phantom tumour, and spurious peritonitis. The author divides cases of meteorism into two varieties, according as the distension is general, affecting the whole abdomen, or localised. In both kinds, the difficulty of diagnosis may be very great. This subject is treated fully and well. As to the mechanism of production of hysterical tympanites, the localised form could be accounted for (Ebstein's theory) by local contractions of the intestinal fibres, whereby local production of gases is favoured, and is imprisoned when formed; or by the contraction of certain of the muscles of the abdominal wall. The generalised variety cannot be so explained. Brodie showed, by introducing a tube into the rectum, that when the gas was passed the abdomen became flattened; it soon, however,

swelled up again. But the tympanites has been known to disappear spontaneously, or under chloroform, without the passage of gas *per os* or *rectum*. Alternating paralyses and contractions of the intestinal walls may partly account for it; but the author thinks it difficult to admit Ebstein's view that there is insufficiency of the pylorus, and that carbonic acid gas and swallowed air passes from the stomach into the intestines. Others have supposed that the gases are derived from the blood, and are re-absorbed by the latter when a sudden disappearance of the distension occurs. In some cases hysterical tympanites simulates intestinal obstruction, even to the presence of fœcal vomiting. In connection with this it may be mentioned that there is evidence that reversed peristaltic action may take place in hysteria, *e.g.*, an enema having been vomited soon after administration.

Hysterical disturbances of the urinary and genital organs receive full discussion. In considering cases of anuria, or ischuria, the author points out that attacks of ischuria of short duration are not uncommon in hysteria, and often pass unnoticed. In the more serious attacks the greater number have for their primary symptom constant vomiting, and the ischuria is merely secondary to this; sometimes, but rarely, ischuria is the primary, and vomiting the secondary affection. The vomitings must be held to be, to some extent, compensatory for the suppression or diminution of urine, as a constant relation is to be observed between the curves of the quantity of fluid vomited and that of urine passed, the one varying inversely as the other. In some cases where anuria or ischuria has occurred with vicarious phenomena, especially vomiting, the patients have gone as long as eight and seventeen days without uræmic accidents supervening. Rossoni has shown in two cases where there was ischuria, and not total anuria, that the administration of twelve grammes of urea in the one, and subcutaneous injection of fifteen grammes in the other, determined an attack of uræmia, though no marked symptoms of uræmia were previously present. The uræmic symptoms were relieved by pilocarpine, which, in the first patient, produced salivation, sweating, and urine containing urea; in the second, sweating and salivation only. The author sums up as follows:—In the large majority of cases hysterical anuria is accompanied by vicarious phenomena, such as vomiting, sweats, or diarrhœa. Cases of prolonged anuria or ischuria without such vicarious symptoms are very rare, and are sometimes accompanied by symptoms of uræmia, on whose gravity, in

the absence of a sufficient number of observations, it is very difficult to pronounce at present.

In the treatment of hysteria it is advised that if the mother suffer from confirmed hysteria, with symptoms of ordinary intensity, the children should be removed from home at an early age, and educated away from it. This is especially important in the case of girls, or, if the children have shown the premonitory symptoms of hysteria in the shape of night terrors, agitated sleep, or somnambulism. Such children are generally precocious, and require no stimulus to intellectual work; they should receive a sound physical training; the love of the marvellous and excess in amusement or in religious exercises should be especially discouraged. The influence of marriage is considered to be, speaking generally, favourable in female hysteria. In pronounced hysteria in men marriage is to be deferred, and the final advice to marry or not must depend on the disappearance or persistence of the hysterical symptoms. There is a useful section on the examination and anamnesis of hysterical patients.

With regard to hypnotism, the author urges the view held at the Salpêtrière. The hypnotic state is considered to be identical with an hysterical paroxysm, but provoked instead of arising spontaneously, and to act like hysterical paroxysms in profoundly modifying "le terrain hystérique." It may, hence, be dangerous even in experienced hands, and should be employed only when the accidents that may be excited by it are less grave than those which it is desired to cure.

It is hardly necessary to say that the importance of isolation in suitable cases is strongly insisted upon. Hydrotherapeutic measures are useful, especially the cold douche. Static electricity is recommended, especially for treatment of anæsthesia and muscular weakness, and as diminishing the frequency of the attacks; it also improves the general state of health and of the digestion, and acts as a tonic. Faradism is especially useful in the treatment of localised anæsthesias.

In the treatment of the convulsive attack, apart from taking precautions to prevent the patient injuring herself, no special measures are advisable. Should, however, laryngeal spasm or other urgent symptoms make it advisable to cut short the attack, the inhalation of chloroform in fractional doses, as in confinements, is the best method to employ. If the attack is the preliminary to a series of fits, or to a comatose state, chloroform is hardly suitable, and hypnotism may then be tried. In lethargic states the author advises to seek for a hysterogenic

zone, whose irritation will throw the patient into a paroxysm, such as is the common natural termination of such states.

In the case of contractures, they are, if possible, to be promptly dealt with; they may be thus prevented in their incipient stage. If practicable, the antagonists of the contractured muscles may be thrown into contraction by stimulation with a faradic current, if anæsthesia be present, or with static electricity, if there is hyperæsthesia. If these measures fail, resort may be had to suggestion during hypnosis. Examination under chloroform of the contracted limb is of great value, in order that if adhesions have formed they may be broken down. When the patient comes round let the limb return to its former position if it tends to do so, M. Charcot's experience having shown that the trouble is only prolonged by fixing the limb in splints in a position opposite to that of the contracture.

In paralysis it is important to prevent contractions of the tendons, &c., arising from a faulty position of the limb; when one limb is paralysed, movements of the sound limb may be usefully employed to call up the dormant sensations of the muscular sense in the paralysed one.

In persistent vomiting, with its consequent anorexia, and in primary anorexia neurosa, isolation is the essential point in treatment; and the author quotes with approval M. Sollier's advice that immediately isolation is established the patient should be made, from the very first, to take ordinary meals. He has found no bad results occur from at once giving the patients solid food; as a rule, they are able to digest it. It should be insisted upon that they eat, and recourse to forced feeding, either by the tube or by means of hypnotism, is undesirable.

The author has consistently endeavoured to make his book a practical clinical treatise, a complete embodiment of all hitherto observed hysterical phenomena. It is thus necessarily of great length, partly owing to the enormous mass of the literature on the subject. This literature, French, German, and English, is very fully treated; French writings largely predominate, but some account will be found of nearly every observation of interest or importance from most sources. Quite a feature of the book, and one which adds greatly to its value as a work of reference, are the historical prefaces to each section, containing much interesting, and often very curious information. The various theories of origin of hysterical symptoms receive only brief notice, the aim of the work being the consideration of hysteria from the point of view of clinical observation, the description of its clinical manifesta-

tions. At the same time the neurosis is treated from the standpoint of the views held with regard to it by Charcot and his pupils, as the outcome of their long continued and laborious investigations on all its phases at the Salpêtrière. The book gives, therefore, a complete statement of what these views are, and, apart from any other considerations, would be interesting and valuable on this ground alone. Whatever the ultimate judgment on these researches may be they must always be remembered with gratitude as having done more than any others to place the study of hysteria on a rational basis, and to elucidate the many obscure problems underlying it.

According to this view hysteria is to be considered as characterised by (*A*) certain fixed signs—the stigmata, consisting chiefly of disturbances of sensation and of the special senses, together with special "diatheses" or tendencies; (1) to contractions "diathesis de contracture," and (2) to muscular weakness, or amyosthenia, the latter in its most developed form finding expression in motor paralyses; and by (*B*) paroxysmal manifestations occurring as episodes on the groundwork of the stigmata of which the most common and most striking are the convulsive seizures. For these seizures a fully developed form is taken and considered to be the type, other paroxysmal disorders representing various phases of the typical attack. Hysteria is treated in this book from this point of view, all its manifestations being referred in their origin to the above characteristics. The plan of the work is thus first to consider generally the characters of the stigmata, in modifications of sensation and motion, then the paroxysmal symptoms, then the chief forms of contracture and paralysis, and, finally, the hysterical affections of the thoracic and abdominal organs, considered as expressions of one or more of the above general characters of the disease in one particular organ.

No doubt clearness is gained by treating a complicated subject on these lines, and the descriptions are simplified and systematised. The recognition of modifications of sensation, and of the special senses (these modifications having more or less peculiar characteristics), as very frequent symptoms, capable of easy identification in cases of hysteria, forms a step in advance in diagnosis. Perhaps the recent researches on the characters of hysterical anæsthesia are especially noteworthy as throwing light on the essential nature of the disease. At the same time the matter is complicated by the readiness with which the hysterical patient reacts to suggestion. Only in this way can the great variations

in the frequency of the occurrence of the stigmata, found by different observers, be accounted for. Such differences are to be partly explained by differences in the methods of examinations, and in the surroundings in which the patient is placed during observation.

A curious fact in the history of hysteria is that whenever a new combination of symptoms is identified as a clinical entity in neurology, cases of hysteria are sooner or later found, in which the newly-described group of symptoms is closely simulated.

It is, however, often difficult to trace out the analogy of symptoms in individual cases with the general type as represented in the above scheme. For instance, it is admitted that the complete attack of "hystero-epilepsy," as given in Charcot's masterly description, is of very rare occurrence, exceedingly so in comparison with the common milder attacks. And the convulsive attack commonly met with bears but a distant resemblance to the supposed complete form. It does not seem that anything is to be gained in simplification, but rather that still further complication is produced by describing the minor attacks in terms of the other. Similarly, in referring symptoms connected with the disturbance of a special organ to a hypothetical general type, assumptions are involved of whose accuracy we have no positive knowledge.

At the same time such a classification of symptoms, regarded as a provisional one, even if in the light of fuller knowledge it may in the future need considerable modification, is a decided aid to the study of the disease, in that it accentuates the fact that in dealing with hysteria we are dealing with a definite group of symptoms which result from a distinct and special disturbance of the central nervous system; and, although extremely diversified, present a certain constancy and order, and do not occur irrespective of all rule. It should be remembered that the investigations of Charcot were the first systematic attempt to bring order out of chaos. The author's observations on the modification of the urinary excreta during the various paroxysmal manifestations of hysteria, based as they are on a long series of carefully conducted investigations, deserve attention, and should be widely carried out. Such a result is of great interest, and if more extended experience confirms it, should prove a valuable aid in the diagnosis of doubtful cases. Mention must finally be made of the many excellent illustrations which adorn the book, and, again, to its value as a work of reference on the subject.

J. Michell Clarke.

The Occipital Lobe and Mental Vision. — J. Soury, *Revue Philosophique*, January, February, December, 1895; February and March, 1896.

(A) From Retina to Primary Sub-cortical Optic Centres.

In these articles, M. Soury summarises the literature of "mental vision" from 1890—when he gave a critical account of its facts and doctrines in *Fonctions du Cerveau* (Paris, 2 *ed.*, 1892). He discusses "(*a*) the cerebral centre of mental vision, (*b*) the intra-cerebral nervous apparatus relative to this function, consisting of primary optic centres (external geniculate bodies, pulvinar of optic thalamus, anterior quadrigeminal tubercles), and the projection and association bundles which unite the retinal fibres to the ganglia of origin for the optic nerve, and these ganglia to the cortex of the occipital lobe." The excitability of the cortex—recent histological methods (Weigert and Pal, Golgi, Ramón y Cajal, Ehrlich, Nissl)—the overthrow of the network theory of Gerlach and Golgi—the establishment of the theory of neurons, and propagation of nerve impulses by contiguity instead of continuity—as well as the comparative and virtually antagonistic development of basal ganglia and cortex of cerebral hemispheres along the scale of vertebrates—form the subject-matter of his critical digest. The following monographs are cited as the most important among the recent publications:—Munk's *Sehsphäre und Augenbewegungen* (Berlin, 1890); Monakow's *Experimentelle u. pathologisch-anatomische Untersuchungen über die optischen Centren u. Bahnen nebst Klinischen Beiträgen zur corticalen Hemianopsie u. Alexie* (Arch. f. Psych., xxiv., 1892); Henschen's *Klinische u. anatomische Beiträge zur Pathologie des Gehirns* (Upsala, 1890-1892); *On the visual path and centre* (Brain, 1893); *Sur les centres optiques cérébraux* (Rev. générale d'ophtalmologie de Dor & E. Meyer, 1894); Wilbrand's papers (*infra*); Sachs on the occipital lobe, *Das Hemisphärenmark des menschlichen Grosshirns. I. Das Hinterhauptlappen* (Leipzig, 1892); Vialet's *Centres cerebraux de la vision et l'appareil nerveux visuel intra-cérébral* (Paris, 1893); and the anatomo-pathological studies of Dr. and Madame Déjerine. (Other references will be found in the bibliography at the end of this abstract).

Constitution of Optic Nerve.

The optic path as a whole runs from retina to cortex, but contains two parallel systems of conductors (*a*) centripetal, (*b*)

centrifugal in direction (R. y Cajal). The former arise in the axis-cylinder prolongations of the large cells of the retina, which form the optic nerve. They arborise freely with the protoplasmic expansions of the principal groups of nerve cells in the external geniculate bodies, and pulvinar of the optic thalamus. From the intercalary cells of these ganglia other axis-cylinder processes traverse the posterior end of the internal capsule, continue as the white sagittal substance, or optic radiations of Gratiolet, and end in the nervous felt-work of the fifth (and third) layer of the occipital cortex. There a new system of neurons (*neurones associatifs*) forms connection with the other cells of the cortex, more particularly with the giant solitary cells.

The centrifugal fibres start with the axis-cylinder processes of the pyramidal cells of the cortex, and arborise with the nerve cells of the superficial grey matter of the anterior quadrigeminal tubercles; the processes from these ganglia again terminate in the nervous felt-work of the retina (Monakow, 1892). The giant solitary cells of the occipital cortex are found to atrophy after section of the posterior segment of the internal capsule. Munk also affirms that centrifugal fibres are directed from the visual area to the centres of movement for the eye.

Reflex and Voluntary Movements of the Eye.

The experiments of Munk, Fritsch, Hitzig, Ferrier, Luciani, Tamburini, Bechterew, and Schäfer are summed up as follows: —(i.) The entire visual centre of one hemisphere is related with the homonymous halves of both retinæ; (ii.) the superior zone of the visual centre of one hemisphere is related with the upper part of the homonymous halves of both retinæ; the inferior zone with the lower part, the intermediate zone with the intermediate part. Schäfer also noted accompanying movements of the upper eyelid and pupillar modifications. Later on he showed that after removal of both frontal lobes anterior to the sylvian fissure, and section of the corpus callosum, electrical stimulation of the cortex still resulted in associated eye movements. The centre of these movements must, therefore, be sub-cortical, and probably lies in the grey matter of the quadrigeminal tubercles.

Danillo also noted conjugate deviation of the eyes, at the fifth month, in puppies and kittens, on exciting the occipital cortex (1888). Excitation of the white matter, after removal of the cortex, produces the same at the second month; equally so

if the cortex of the anterior motor region is removed, or isolated by vertical or horizontal sections. Excitation of the white matter of the motor zone produces no movement of the eyes. From this Danillo concluded that—(i.) The occipital lobes do not comprise centres analogous to those of the motor zone; (ii.) Ferrier's hypothesis (which accounts for the movements of the eye by subjective optic sensations, due to excitation of the occipital lobe) cannot be sustained; since stimulation of the white matter provokes the same movements, both after ablation of the grey matter, and at a period when the cortex is still inexcitable; (iii.) the phenomena observed after vertical and horizontal section of the brain, or extirpation of the motor zone, indicate that the centres of conjugate deviation lie deeper than either motor region of the cortex or occipital lobe. M. Soury suggests that the electrical stimulus in these cases may act upon preformed cerebral mechanisms, replacing the physiological nervous stimulus.

Munk established similar conclusions as to the independence of the eye movements caused by excitation of the occipital lobe; and since a horizontal incision, dividing the fibres of the corona radiata of the visual sphere, invariably blocked them, it is obvious that the oculo-motor functions of the occipital lobe are in relation with the basal ganglia *viâ* the optic radiations of Gratiolet. The occipital lobe can, therefore, no longer be regarded as purely sensory, seeing that its excitation provokes movements of the eye, upper eyelid, and iris. In contradistinction to the oculo-motor effects of visual sensation, due to excitation of the visual sphere, Munk determines other movements, which persist after ablation of both cortical centres of vision. He distinguishes (i.) *retinal reflexes*, or contraction of the pupil to light, even after removal of the cortex; (ii.) *visual reflexes*, *e.g.*, winking, avoidance of obstacles; (iii.) *acquired visual reflexes*, *e.g.*, flight from a whip, &c. Excitation of the occipital lobe induces reflexes of the second order. "We know," continues Munk, "that the corona radiata of the visual sphere contains, besides the fibres of the optic nerve, whose excitation, running centripetally to the visual area, determines vision, other fibres travelling in the same radiations, of which the excitation—starting with the visual sphere and passing centrifugally to the lower, sub-cortical regions of the brain—determines movement; and that the simple movements of the eye (as well as the movements of the eyelids, &c.), resulting from vision, are effected by this path; while other movements contingent upon sight need the inter-

polation of association-bundles and other cortical elements." These movements of the eye can, therefore, only be due to centrifugal impulses from occipital lobe to sub-cortical centres, *viâ* corona radiata (Sherrington, 39). Monakow places these centres in the anterior quadrigeminal tubercles. Knies further refers the movements of convergence and accommodation, which he characterises as "voluntary," to the occipital lobe; so that its cortex is both sensory and motor. And, in fact, convergence and accommodation are affected in all cortical troubles of vision, while the inferior ("involuntary") ocular reflexes may persist.

Clinical evidence shows that lesion of the anterior quadrigeminal tubercles is not essentially injurious to vision, but that it does involve disturbance of eye-movements and of pupillar innervation (Monakow). In the lower animals, even in certain mammals, these ganglia are of main importance to vision, as compared with the external geniculate bodies and pulvinar of the thalamus. The fibres which they contribute in man to the optic tract are relatively insignificant (Monakow, Bernheimer). The *pupillar luminous reflex*, however, appears to be governed by these bodies.

Argyll Robertson was the first to show (1869) that this reflex (direct contractility to light) can be abolished; while movements of accommodation and convergence are preserved. The fibres of the optic nerve transmit the luminous stimulus which governs movements of the pupils, to the point at which originate the branches from the oculo-motor nerve that innervate the sphincter of the iris. Wernicke finds that the reflex arc between retina and oculo-motor nerve, does not extend beyond the quadrigeminal tubercles, since (i.) when the path of the pupillar fibres is interrupted behind these ganglia, the reflex arc is left intact, and the pupils give nominal reaction to light; (ii.) when it is interrupted in front of them, the pupillar reflex fails to appear. Mendel's detailed experiments point to the *ganglion habenulæ* as the precise centre of the reflex. Iridectomy seems invariably to produce atrophy of this organ. He, accordingly, defines the path of the reflex as optic nerve and ganglia of habenula on same side, thence by posterior commissure to nucleus of Gudden, and so to fibres of oculo-motor trunk. It is to be noted that the loss of the pupillar reflex occurs in fifty to sixty per cent. cases of general paralysis, in which lesion of the ventricles, more especially of the third, is a common feature. The expression of the emotions shows the same anatomical and functional independence, accord-

ing as the facial movements are reflex or voluntary in origin. In the latter, the nervous impulses travel *viâ* internal capsule, and the foot of the cerebral peduncle; in the former, *viâ* optic thalamus and tegmentum of the peduncle. The loss of the emotional involuntary reflex of the facial nerve depends upon lesion of a sub-cortical ganglion (optic thalamus, or fibres of corona radiata); voluntary movements, such as mimiery of the emotions, are inhibited by lesions of the cortical centre of the same nerve.

Functions of Cerebral Cortex in the Scale of Vertebrates.

Passing on to the deductions made by Edinger (10), and by Goltz himself (15), from the effects of Goltz's operations on the cerebrum, M. Soury denies that any luminous sensation, or perception, can exist after the cortex has been totally extirpated. He refers the facts adduced to survival of sub-cortical centres, and to reflex actions usually associated with visual sensation, but intrinsically independent of it. Such are the pupillar reflex, and the *Blendungsreflex*, cited by Goltz.

Edinger's preparations plainly show the lacuna which exists between the dog's brain and that of man, or even the monkey. In the latter, minimal destruction of the Rolandic cortex determines paresis or paralysis—impotence of voluntary movement, and troubles of sensibility; in the dog (as in lower vertebrates) loss of the cortex does not imply loss of co-ordinated movements, nor of such as are voluntary or acquired, *i.e.*, represented in the cerebral cortex. Soury further pleads that Goltz's dogs are in a state analogous to the dementia of paralysis, and are, therefore, of no account in the *pros* and *cons* of cerebral localisation. In man, he concludes, (as opposed to lower vertebrates), total destructive lesion of the cortex of both occipital lobes produces absolute cecity: man cannot see with his lower, sub-cortical optic centres.

Trophic Centres of Optic Nerves.

The direct trophic influence of the retina upon the optic nerves is well-established. In cases of anopthalmia, the optic nerves, chiasma, and fillet, are wanting, the occipital convolutions on either side being atrophied. In subterranean animals, whose visual apparatus has degenerated, the tactile sense is peculiarly responsive to luminous excitation. Dubois (7) reduces the mechanism of vision, in the last resort, to a tactile manifesta-

tion. The syphon of pholas dactylus contracts to light, its photodermic functions resembling those of the retina.

Connection of Retina with Primary Optic Centres.

The histology of the retina and visual fibres are considered under this heading, but must here be omitted. The visual acuity of the *macula lutea* is said to depend upon the delicacy with which each cone (7,000 to 7,500) transmits its excitation to a bi-polar cell, this again to one protoplasmic arborisation of the ganglionic layer. The autonomy of the macular region persists throughout the optic nerve to the cortical centres; and the preservation of central vision in the majority of cases, where (as in hemianopsia) the entire visual cortex is destroyed on one side, indicates that each macula is connected with both hemispheres. The line which separates blind half from normal half of the field is not median; it passes five to ten degrees beyond the centre, leaving the macular region intact.

The importance of the sub-cortical centres must be remembered. They are the ganglia of origin for the optic nerve—the post-house where impressions conveyed by the retinal fibres are transmitted to other couriers. The transmission of visual impressions must be considerably modified by the distribution of the retinal fibres in the basal ganglia. An axis-cylinder process may interlace with one or with several dendrites of the nervous layer, which again reacts upon the cortical distribution.

In re centrifugal fibres, M. Soury adduces, as physiological evidence of their existence in the optic nerve, the discovery of Engelmann (11) of the movements of the cones and pigment of the retina under luminous and nervous influences. The cones of the eye retract in light. In a "dark" frog, direct illumination of one eye causes the other to contract. When the brain has been destroyed, light only affects the eye directly illuminated. This functional sympathy between the two retinæ can only be explained by synergic association of the cones through the optic nerves, acting centrifugally instead of centripetally. Hence Engelmann's hypothesis of two kinds of fibres in the optic nerve—(*a*) sensory, centripetal, conducting luminous and chromatic sensations; (*b*) centrifugal, motor, or "retino-motor." Indirect excitation of the optic nerve by strychnin produces the same action on the retina. Additional evidence is contributed by Nahmmacher's experiments on amphibia (1893), Gotch and Horsley's on the spinal cord (1892.)

(B) From Primary Optic Centres to Cortex of Occipital Lobe. Calcarine Region and Cortical Retina.

M. Soury adopts the position of Henschen in regard to the cortical centre of vision. "Le lobe occipital est le centre cérébral de la vision mentale. De la rétine aux centres primaires optiques —corps genouillé externe, pulvinar de la couche optique, tubercules quadrijumeaux antérieurs—et des centres primaires optiques à l'écorce du lobe occipital, des faisceaux de projection relient les fibres rétiniennes,—nerfs optiques, chiasma, tractus,—aux ganglions d'origine des irradiations optiques, dont les fibres se terminent par des arborisations libres sur la face interne du lobe occipital, dans le territoire de la scissure calcarine, véritable rétine corticale." The visual area is much more extended than the centres for perception of sensations of light and colour; and very different theories obtain as to the functions of those regions of the occipital and parietal lobe which it comprises. The anatomo-clinical method (Monakow, Henschen, Déjerine, Sachs, Vialet, Redlich) throws more light on the functions of the nerve centres than the earlier anatomical or purely experimental researches. Thus it has been shown that in cases of blindness, involving widespread degenerations, the geniculate bodies alone are in direct relation with vision. The fibres of the optic nerve which pass into the pulvinar of the thalamus, the anterior quadrigeminal tubercles, the temporal and parietal lobes, are not visual (Henschen).

Degeneration of the pulvinar does not produce heminanopsia, when the geniculate body is intact. The anterior quadrigeminal tubercles may submit to grave lesion without disturbance of vision. The optic fibres in these ganglia must be accessory to the mechanism of vision; they do not conduct luminous and chromatic impressions from retina to cortex. The pulvinar and quadrigeminal tubercles are possibly reflex optic centres—in man they probably do not contain a single visual fibre. The geniculate bodies alone are in relation with vision, and lesion of these ganglia inevitably causes hemianopsia. Certain forms of sub-cortical hemianopsia may indeed be accompanied by pulvinary lesion, but the actual cause of cecity is lesion of the external geniculate body. Destruction of this ganglion determines an almost total disappearance of the bundle of Gratiolet, through which pass the visual fibres from the ganglion. So, too, in retrograde degeneration, the partial destruction of the occipital lobe may determine atrophy of the internal capsule, yet the fibres

involved are not visual. Lesions of the occipital lobe react upon the retro-lenticular region of the internal capsule—the fibres of which end in the central grey nuclei, while the capsular fibres proper pass on into the cerebral peduncle. (Déjerine.)

Clinical application of these conclusions is not slow to follow. Pick recognises that a lesion exactly limited to the optic thalamus produces no alteration in perception of colour and form, nor any deficit in the visual field. Lesion of the external geniculate body, or optic radiations, is required to provoke these troubles. The symptoms which originate from the thalamus are quite different, *e.g.*, motor troubles of expression—imitative, involuntary, automatic, &c. In man, the anterior quadrigeminal tubercles are equally deprived of the importance which Griesinger attributed to them in vision. Recent pathological observations prove that lesions of these ganglia may develop without essential alteration of vision; they involve motor troubles of the eye, and of pupillar innervation. The optic lobe in man is no more than a reflex centre. As we have seen, it is still (without speaking of the luminous pupillar reflex) of prime importance in the transmission of centrifugal impulses (*supra*, Wernicke, Monakow, R. y Cajal).

Nor has the inferior parietal lobule, or angular gyrus, any more direct relation with vision. Ferrier's localisation of the cortical centre of distinct vision in the angular gyrus of the opposite hemisphere—Charcot's explanation of the crossed unilateral amblyopia of hysterical hemianæsthesia, by a complimentary decussation of the direct bundle of optic fibres, are, therefore, of no more value than the early observations of Goltz, Ferrier, and Munk, to the effect that unilateral lesion of the visual centre in dog and monkey determines complete cecity of the opposite eye, and not partial cecity of both retinæ—a conclusion based on the theory of complete decussation in these animals.

It was reserved for Henschen[1] to point out the true analytic method. "When the visual area is removed, the fibres which conduct the luminous impressions degenerate; secondary changes also occur in the fibres which unite the visual area with other portions of the cortex and with the central ganglia." In a case of hemianopsia, *e.g.*, it is illegitimate to charge with the visual disturbance all tracts that are found degenerated. Negative must be opposed to positive cases, and the principle of elimination, as laid down by Exner, Séguin, &c.,

[1] Henschen. "Sur les centres optiques cérébraux," p. 3, Extrait de la *Rev. génér. d'Ophtalmologie*, No. 8, 1894.

must ultimately result in the discovery of one constant, invariable lesion in cecity. Henschen,[1] by careful clinical comparison, arrived at the *cas type*, and determined the inevitable lesion to be localised in the cortex of the calcarine fissure. The other lesions observed have only produced blindness, inasmuch as they involved the visual fibres of the radiation of Gratiolet on its way to the calcarine cortex. The optic centre cannot, therefore, be localised on the external surface, or convexity, of the lobe—as determined by Munk and other physiologists: such lesions as they diagnosed must have involved the optic radiations. In Henschen's words, "hemianopsia results from a lesion only when it destroys the calcarine cortex, or the optic bundle which unites the geniculate body to this part of the occipital lobe. This bundle occupies the inferior part of the radiation of Gratiolet."

In Henschen's typical case, in which the patient was an intelligent subject, not suffering from dementia, the lesion was a softening produced by thrombosis, strictly confined to the cortex, lying deep in the calcarine fissure, and not involving more than one or two mm. in depth of the subjacent white substance. The case was not complicated by any lesion of the central ganglia. Microscopic investigation only revealed a secondary degeneration in the optic radiations, in consequence of the cortical softening.

The anatomical structure of the calcarine cortex (though differing from the rest of the occipital lobe, not merely in the thickness of the molecular layer, but also in its development of horizontal fibres, forming the band of Vicq d'Azyr), is not special to the fundus of the fissure. The same band extends for several mm. along the two lips of the calcarine fissure, the superior of which belongs to the cuneate, the inferior to the lingual lobe. It is to be expected that such identity of structure in either convolution should result in identity of function. And Hun adduces a case in which atrophy of the superior lip of the calcarine fissure produced hemianopsia of the inferior quadrant of the visual field on both sides. Wilbrand shows that the lower lip corresponds with the upper visual field. There would thus appear to be projection of the retina upon the cortex of the occipital lobe, as contended by Munk, and disputed by Monakow. Henschen accepts it, and proposes the name of "calcarine retina" for that part of the cortex of this fissure at which, according to him, there is projection of the elements of the peripheral retina, *viâ* the visual fibres of the optic nerves, chiasma, tracts, and optic radia-

[1] Cas de Henschen-Nordensen. "Klin. u. anat. Beiträge zur Pathologie des Gehirns," II., p. 387. Obs. XL.

tions. The macular bundle, though lateral in the papilla of the optic nerve, is certainly central in the chiasma and tract. Beyond the external geniculate body, however, Monakow's anatomical objections appear to be well-grounded. Vialet proposes a physiological shortening of the path of the visual impulses, due to association, so that a ganglion cell of the retina might always be in relation with the same cell, or group of cells, of the cortex, owing to the incessant repetition of the transmitted impressions. The central and peripheral parts of the cortical retina would lie, according to Henschen, in the anterior and posterior parts of the cortex of the calcarine fissure. Sachs, again, has a case which does not correspond with this somewhat restricted cerebral localisation of the *macula lutea*. Henschen, like Wilbrand, admits that each half of either *macula lutea* is in connection with both cerebral hemispheres, while the macular fibres also undergo partial decussation in the chiasma, and are divided into direct and crossed bundles.

The preceding observations refer to the "cerebral retina," and not to the much larger region of the "visual area," which is the anatomical substrate of mental vision. While the region on which visual impressions are projected is very limited, that corresponding with visual representations is vast, and may very likely involve the convexity of the occipital lobe and the angular gyrus. Henschen concludes that the seat of perception and of representation occupy distinct positions on the occipital and parietal lobes. Vialet distinguishes "a visual centre of perception" and "a visual centre of memory," explaining their relation by association bundles. Déjerine localises the "visual centre of words" in the angular gyrus.

Nothing is certain, however, beyond the fact that there is a cortical centre of visual perception, and that its lesion entails cortical hemianopsia. As to psychical cecity there are no proofs; it is a question of interpretation. At this point, Munk's dual schema of the cortical elements of perception and representation gains general acceptance, but simply as a *pis-aller*, because no other explanation is to hand, *e.g.*, in cases where the patient suffers from verbal cecity, loss of intelligence of written signs, along with unimpaired vision of the same signs. It is thus easy to pile up distinct centres of memory for all external objects. Vialet regards it as certain that some cerebral elements gather up new perceptions, while others warehouse the visual memories. M. Soury urges, on the contrary, a more sceptical attitude until further clinical evidence shall be forthcoming.

He sums up by defining the "optic path," or system of visual fibres properly so called, as constituted by two neurons—(1) an *anterior neuron*, formed by the great ganglion cells of the retinæ and their prolongations, the elements of the bundles of the optic nerve, the chiasma and optic tract, the terminal axis-cylinder ramifications, arborising in the protoplasmic branches of the cells of the external geniculate body; (2) a *posterior neuron*, formed of the cells of the external geniculate body and their prolongations, which, as fibres of the inferior portion of the optic radiations, arborise in the middle of the different layers of nerve cells in the cortex of fundus and lips of the calcarine fissure.

The Occipital Lobe.

Space forbids us to enter fully upon M. Soury's review of the comparative morphology, arterial supply, embryology, and histology of the occipital lobe. This lobe is small in man as compared with the rest of the hemisphere; in monkeys it is highly developed. On the other hand, it is in man (unlike the monkey) as highly convoluted as the rest of the cerebrum; hence the superior size of its surface, no doubt in relation with the increased number of fibres from the optic radiations which spread over the calcarine region, and effect a high elaboration of central visual impressions. The external convex surface presents no connecting gyri between the occipital and the parietal and temporal lobes. So, too, the internal surface is continuous with the temporal lobe. The external parieto-occipital fissure is very small. In microcephalous cases (= pure arrest of development — idiocy) it is extensive; while the external connecting gyri become once more pronounced, and the occipital lobe is smooth, recalling that of inferior monkeys.

M. Soury does not admit the transverse occipital sulcus to be the homologue of the "*sulcus du singe*," as held by Ecker and Rudinger. In man this sulcus divides the occipital lobe into the superior and inferior regions. Where the "*sulcus du singe*" really exists, it seems, again, to imply arrest of development.

On the internal surface the calcarine fissure appears in the first months of intra-uterine life as two branches, which subsequently unite with the parieto-occipital fissure to enclose the cuneus. (In micro-cephalics the cuneate lobe is small and smooth, while the *pes hippocampi minor* is often wanting. In

Chinese brains, the cuneus appears to be very small). The calcarine fissure above, and the collateral fissure below, enclose the lingual lobe. Between the collateral sulcus and third temporal fissure lies the fusiform lobe. The cuneus, lingual lobe, and fusiform lobule are the three convolutions o which the lesions appear to account for hemianoptic symptoms, and which many clinical authorities regard as the cortical centre of vision.

These regions of the brain are supplied by the posterior cerebral artery; in particular by its two posterior branches, the posterior temporal and occipital arteries. The latter ramifies over the calcarine fissure, and, under the name of calcarine artery, is responsible for the nutrition of the most important regions of central vision. Monakow remarks that the obliteration of this artery is especially provocative of lesion, because it induces softening of the cuneus. The occipital artery may thus be termed artery of cerebral vision, or hemianoptic artery. The obliteration of any one of its branches, in particular of the calcarine artery, induces a lesion (anatomical and functional) of the cuneus and surrounding regions, especially of the lingual lobe. In arterial thrombosis, the seat of the softening is often less extensive in the cortex than the vascular region; because there is a possibility of collateral irrigation from neighbouring vessels, since the cortical arteries are not terminal, and anastomose freely (Mendel). In the white substance and basal ganglia, on the contrary, the arteries are terminal. Monakow insists on the gravity and complexity of troubles in the visual apparatus, arising from even a restricted area of softening of the white matter of the cuneus, since this involves the destruction of projection fibres passing from the primary optic centres of the convolutions of the convexity and lingual gyrus. It is more especially in regard to vascularisation in the central nervous system that the connections of its respective parts are of such importance; for the destruction of a nerve centre by anæmic softening or hæmorrhage, or the mere slowing-down and progressive extinction of its activity by arterial obliteration (local anæmia), reacts infallibly on the regions of the brain connected with this centre. The innumerable neurons which come from all points to terminate here, whether by their collaterals or by the arborisations of their axis-cylinders, as well as the dendrites or cellular bodies of the neurons of the affected centre, undergo, in consequence of the disturbance of their normal activity, alterations in nutrition, so profound as often

to reduce to silence considerable, and sometimes distant, regions of the cerebral cortex. M. Soury queries the use of the term functional lesion (in cases where the original lesion cannot be detected on examination of the cortex); all functional alteration may, in the last resort, be reduced to a disturbance of nutrition, taking nutrition to mean the most general cause of modification in the structure and texture of the anatomical elements. He contends that the key to an interpretation of the functions of the occipital lobe—*i.e.*, to an understanding of mental vision—lies in the anatomy of its white matter, and connections with the neighbouring regions of the parietal and temporal lobes. The anatomical and functional relations of these lobes are of the utmost importance to the study of optic aphasia, verbal cecity, and alexia. In the anatomical descriptions that follow, Sachs (36) and Vialet (43) are the principal authorities quoted.

In describing the *forceps corporis callosi*, Sachs postulates the existence of at least two nerve paths. If the cortical region of distinct vision in both eyes, corresponding with the macular region of the retinæ, is represented in the two occipital lobes, the two cortical points of distinct vision must be commissured by the fibres passing through the forceps. Again, the right occipital lobe must be associated directly with the left temporal lobe, in order that objects seen in the right half of the visual field may evoke the corresponding auditory verbal image localised in the left temporal lobe. This path (according to Freund) must be interrupted in optic aphasia.

In the histology of the occipital lobe, Soury adopts the 6 cortical layers of Ramón y Cajal (as against Meynert's 8). These are—(1) molecular layer; (2) layer of fusiform vertical cells; (3) layer of median myeline cells, or striated band of Vicq d'Azyr; (4) middle pyramidal layer; (5) giant pyramidal layer; (6) layer of polymorphous corpuscles.

A schema of the visual path to the occipital lobe (after Monakow) again presents the idea of retina, basal ganglia, and occipital cortex as optic centres for the departure and terminus of systems of optic fibres—"systems that are highly solidarified, since disturbances of nutrition, defective lesions, react from cortex upon primary optic centres, and from these centres upon the cortex." The schema accordingly includes two sensory and two motor neurons, with the cortical association cells of the external molecular layer.

In this layer (as elsewhere in the cortex) are the giant pyramidal cells, which receive the impact of nervous commotion,

and ultimately (by the process of muscular adjustment) adapt the organism to its environment—internal to external conditions. Nervous impulses pass to these cells from (1) cells of Golgi, with short axis-cylinders; (2) association cells between the several lobules of one hemisphere; (3) cells of the opposite hemisphere *viâ* callosal fibres; (4) sensory (visual) cells; (5) cells of different layers of the cortex, arborising in the molecular layer.

The same molecular layer gives origin to "the motor reactions, still occasionally termed *voluntary*." M. Soury points out that this word no more explains these reactions than does *consciousness* explain the vital processes of nerve-cells that have reached a certain intensity and duration. The nervous discharge, of internal or external origin (produced by the relative intensity of one group, or several groups, of associated mental images, or from the application to some region of the cerebral cortex of mechanical, chemical, or electrical stimuli), provokes, or arrests, muscular contractions of corresponding magnitude or intensity—actions or inhibitions, according to the nature of the centres excited. In sleep, or in chloroform narcosis, the motor reaction follows fatally the nerve-paths of least resistance, most worn, most frequently traversed; and the effect thus determined is inevitable. In narcosis the reaction is certainly unconscious; it is more or less conscious in dreams; in the waking state it is altogether conscious, though it varies with the nature of the act. But this epiphenomenon, consciousness, can no more modify the arrest, or production, of phenomena of central innervation, than the shadow of the traveller alters the rhythm or direction of his steps. The impulses transmitted from the sensory neurons to the dendrites of the great pyramidal cells of the molecular layer of the cortex, are the effective incitation of voluntary movement (R. y Cajal). In physiological excitation of the cortex, the stimulus may act directly on the dendrites of the pyramidal cells, or indirectly *viâ* nervous fibrils from the superficial layer of the cortex. In either case the artificial stimulus of physiological experiment acts on the highest nerve centres of the brain exactly like "the will."

Hemianopsia.

The characteristic symptom of lesions of the cortical centre of vision is homonymous, bilateral hemianopsia. Lesions of the right occipital lobe abolish function in the right half of each retina; the left half of the field of vision is blotted out; and *vice versâ*. The sense of light or of colour may be affected. Hemidyschromatopsia (loss of perception of some colours), or hemi-

achromatopsia (loss of all colours) may precede hemianopsia in the halves of the visual field affected. In ophthalmic migraine, where the transitory disturbances (hemianopsia, cortical cecity, aphasia, anæsthesia, paresis, &c.) appear to be determined by equally transitory states of anæmia in the occipital and temporal lobes, the loss of colour-sense may precede the loss of perception of light and space. The transitory scotoma of ophthalmic hemicrania is a central disturbance (hallucinatory) from the cortical centre of vision, although the patient "sees black," a peculiarity formerly supposed to distinguish sub-cortical from cortical hemianopsia (Mauthner, Dufour).

Lesion of the internal surface of the occipital lobe inevitably produces cecity in the corresponding half of both retinæ, *i.e.*, in both eyes, never a crossed, monocular cecity of the opposite eye (Wilbrand, 1881). Ferrier is, therefore, disallowed (when he accounts for crossed amblyopia by saying that if the occipital lobe is in relation with the homonymous halves of both retinæ, the angular gyrus is more particularly in relation with the yellow spot of the opposite eye), and is rendered responsible for all later errors with reference to the functions of the angular gyrus, crossed amblyopia, &c., of both French and English writers. Nor was Charcot more successful in explaining the crossed cecity of hysterical hemianæsthesia. He assumed a second decussation of the direct bundles of the optic fillet (that did not cross in the chiasma), so that the optic nerve would cross over completely before entering the brain, whereby the unilateral lesion of one hemisphere would produce cecity of the opposite eye. His other theory, that in common sensory anæsthesia, the special sensory anæsthesia which is its usual concomitant in hysteria and other neuroses, as well as in certain organic affections of the brain, derives from lesion of the posterior segment of the internal capsule, is also invalidated on anatomical grounds (Bechterew).

Charcot's clinical facts (as gathered up by Féré, 12) remain for explanation. They may be summarised as follows:—

(1) The intensity of the amblyopia is always proportional to the intensity of anæsthesia in the eye; it is less pronounced when the conjunctiva alone is insensible; more pronounced when the cornea is equally anæsthetic.

(2) If the hemianæsthesia merely affects the extremities and leaves the face intact, the visual disturbance may be absent; but if the face is affected and the extremities intact, there will be amblyopia.

(3) If in hysterical hemianæsthesia, the hemianæsthesia can be suppressed, the amblyopia disappears also.

The same facts apply to the crossed amblyopia consequent on organic lesions of the brain.

Bechterew notes the relation between general anæsthesia and weakness of sight on the same side, or common and special sensory anæsthesia, and extends it even to hypnosis. Frankl-Hochwart noted that in hysteria, unaccompanied by troubles of sensibility, the visual field was usually normal, together with visual acuteness and perception of colour (which, however, might be slightly diminished). It is especially in anæsthetic hysteria that the visual field is concentrically reduced, and the perception of light and colours lowered. There is an evident relation between functional troubles of the eye and disturbances of its sensibility. Knies, in his "Troubles centraux unilatéraux de la vision dans l'hystérie," described the troubles that accompany general hemianæsthesia, including the eye and its annexes (cornea, conjunctiva, &c.), as a diminution of central visual acuteness, nearly always implying concentric reduction of the visual field, and alteration of chromatic sensibility; the functions of the eye being normal, and the state of the pupils variable, with no known corresponding anatomical lesion. These troubles are subject to modification by psychical influences. The chief objection to a cerebral origin of such unilateral troubles is anatomical. Above the chiasma, no lesion of conductors or optic centres is known that can determine unilateral visual troubles. Hence the long acceptance of Charcot's posterior chiasma. Knies offers a new hypothesis: the proximate cause of these unilateral troubles of vision in hysteria is to be localised in the peripheral optic apparatus; the effective cause is central—*viz.*, a cerebral disturbance of vascular innervation.

All brain activity is accompanied by vaso-motor processes, which are vaso-constrictive in nature. The ablation of a cerebral hemisphere is followed *inter alia* by hemilateral paralysis of the cervical sympathetic. In hysteria the loss of the vaso-constrictive action, exercised normally by the centrifugal nerve impulses of the cortex, determines vaso-dilatation of certain peripheral regions. The calibre of the vessels enlarges, and since this occurs at the *foramen opticum*, the nerves undergo mechanical compression. This is translated into disturbance or transitory abolition of condition, and of the function of the sensory organ connected. Such compression of the myeline of a nerve may produce abolition of its conductivity without destroying the axis-cylinder. When the vascular dilatation ceases, the effect, *i.e.*, the functional trouble, disappears also. Hysteria would, therefore, according to Knies,

be "a disturbance of vaso-motor innervation, of cerebral origin." Many other hysterical symptoms can be explained from the same cause, as well as hypnosis, suggestion, sleep.

Bechterew concluded that amblyopia depends upon anæsthesia of the ocular capsule. Section of the sensory or ascending root of the trigeminal (which induces anæsthesia to touch and pain in half the face and head, with amblyopia of the anæsthetic side, and diminution of sensibility in other sense organs on the same side), proved that amblyopia can be induced by lesion of a sensory nerve, of which the roots are distributed to the face, and more especially to the ocular capsule. In other organs, too, diminution of function is related with anæsthesia of the surface. This is the explanation of amblyopia, and sensory anæsthesia in general, in cases of unilateral anæsthesia of the face and sense-organs of cerebral origin—at least, in cases of lesion of the cortex and posterior third of the internal capsule, provided the special conductors to the sense-organs (optic nerve, acoustic nerve, &c.), and their cortical and sub-cortical centres, are not affected.

The detrimental influence of facial anæsthesia on the sense-organs arises from troubles of nutrition. The nutrition of any organ is imperfect unless the sensory and motor nerves distributed to it, and especially the vaso-motor nerves, are in a normal state.

Any interference, central or peripheral, produces the same effect. Duval and Laborde proved that lesion of the ascending root of the trigeminal affects nutrition in the eye; Lannegrace shows that cerebral lesion produces a similar disturbance. All interference with nutrition in a peripheral sense-organ is translated into a corresponding functional alteration, *i.e.*, alteration of sensation; and Bechterew refers such disturbances of nutrition to inadequate arterial irrigation. Hence the capital importance of the state of the vaso-motor system to the interpretation of amblyopia, and of sensory anæsthesia in general, in sensory hemi-anæsthesia.

The acuteness of perception, far from diminishing, may be augmented, if the afflux of blood is considerable in the organs of the special senses, or in surfaces of the skin and mucosæ serving for the perception of tactile, painful, or other excitations. We know that contraction of the vessels from cold diminishes the perception of cutaneous excitation to the point of anæsthesia, while it rises to hyperæsthesia under the action of causes which dilate the vessels (heat, sinapism, &c.). Sensations of cold, absence of sweat, analgesia, defective hæmorrhage after profound pricks in

hysteria, are, like anæsthesia, explained by the diminution of the finest arterial vessels of the surface of the skin. The reality of this vascular spasm in hysterical hemianæsthesia is attested by the loss of heat on the insensitive side of the body, by the greater resistance offered to the electrical current, &c. In artificial anæsthesia, centrally provoked, the cutaneous regions rendered insensitive are lower in temperature than the surrounding regions.

Anæsthesia of the skin and mucosæ produces retinal anæsthesia, amblyopia of the eye on the corresponding side, by depressing the nutrition of the anatomical elements which receive external stimuli of luminous or chromatic sensations, and this solely in virtue of vaso-motor troubles, caused by inadequate irrigation of the peripheral organ of vision. Bechterew's theory[1] (which is extended to other sense organs) explains the crossed amblyopia of symptomatic hemianæsthesia in certain neuroses, or organic affections of the brain, without implicating the optic conductors, primary optic centres, or calcarine region of the occipital lobe. Disturbance of vaso-motor innervation, anæmia of the peripheral organ of vision (in consequence of cutaneous anæsthesia extending to this organ, as to other sense organs), is the cause of sensory anæsthesia, and it is superfluous to invoke either Charcot's "*carrefour sensitif*," or the equally erroneous hypothesis of a complementary decussation of the direct bundles of the optic fillets, behind or within the quadrigeminal tubercles.

Central hemianopsia is never monocular. Monocular hemianopsia, temporal or nasal, may result from lesion due to compression of the internal or external sides of the visual fibres of the optic nerve, previous to their partial decussation in the chiasma. This lesion involves the pupillar fibres, and the pupil of the corresponding eye reacts feebly or not at all to direct illumination, while it reacts synergically to illumination of the other non-affected eye, since the reflex paths are intact on the side of the uninjured nerve, as well as the centrifugal path from the reflex centres to the iris of the affected eye—this is the consensual reaction of both pupils. The pupillar fibres, which originate in the retinæ, are distinct from the visual fibres in the optic nerve and chiasma, while they are not to be found in the occipital optic radiations. It is a moot point whether they pass into the optic tract. The pupillar reflex to light persists in

[1] Bechterew. "Ueb. die Wechsel-beziehung zwischen der gewöhnlichen u. sensoriellen Anästhesie (Functionsabnahme der Sinnesorgane) auf Grund klinischer u. experimenteller Daten." *Neurol. Centralbl.*, 1894.

total, or cortical, cecity, *i.e.*, double or bilateral hemianopsia, due to lesions of both occipital lobes. Henschen finds pupillar fibres in the optic tract up to the level of the external geniculate body, which they do not enter; the hemiopic pupillar reaction is not therefore provoked by lesion of the external geniculate body. Bechterew, on the other hand, holds that the pupillar fibres diverge from the tractus as early as the chiasma, to enter the wall of the third ventricle.[1] The crossed cecity determined in birds by the destruction of one of the optic lobes, leaves intact the contractility to light of the pupil of the blind eye. Before entering the optic lobes with the visual fibres, the pupillar fibres, therefore, divide off from the tractus, and go directly to the region of the nuclei of the 3rd nerve. Bechterew found that both pupils reacted to light after section of the optic tract, in mammals. In man, he affirms the persistence of the pupillar luminous reflex in cases of cecity due to destruction of the quadrigeminal tubercles by compression from a tumour of the pineal gland. In general paralysis and tabes, the pupil may have lost the luminous reflex, while preserving the reflexes of accommodation and convergence, neither vision nor movements of the eye having undergone any alteration. The pupillar fibres have thus from a certain point a separate path from the visual fibres, from which they are throughout absolutely distinct. Darkschewitsch and Mendel, Edinger, Bechterew, Flechsig, Bogrow, have as many theories as to this path. Nothing seems certain except that two bundles of fibres pass from the optic tracts to the central grey matter of the third ventricle, and may be in relation with the nuclei of the ocular-motor nerve.

Wernicke's hemiopic pupillar reaction comes under the category of hemianopsia. Pick gives the reflex arc of the luminous pupillar reflex as composed of a *reflex centre* (no doubt localised in nuclei of ocular-motor nerve), and a *centripetal path* (optic nerve, chiasma, tract, quadrigeminal tubercles, and nuclei of the 3rd pair). If the pupillar fibres, which effect the luminous reflex of the pupil, are simultaneously attacked on the homonymous sides of both retinæ, by lesion of the visual fibres of the optic tract, and the two anæsthetic halves of the retinæ are illuminated (avoiding diffusion of light), the pupils will not contract; if the light falls on the sensitive halves of the retinæ, the pupillar reflex comes off. This is the hemiopic pupillar reaction.

[1] *Ib.* "Ueb. Pupillenverengernde Fasern." *Neurol. Centralbl.*, 1894, p. 802.

By compressing the chiasma at different points it is possible to produce *bi-temporal hemianopsia*—loss of function in the crossed optic bundle; *nasal hemianopsia*—lesion of the external or direct bundle; *superior and inferior hemianopsia*—compression above or below. Atrophy of the visual fibres by compression of the chiasma may also result from internal hydrocephalus.

Pierre Gratiolet, in 1854, discovered the central origin of the optic nerve. Meynert accepted his conclusions. Wernicke worked them out *in extenso*. Panizza, 1855, also pointed out that the bundles of fibres issuing from the posterior cerebral convolutions contributed to form the optic nerve, but he was less acute in his observations than Gratiolet; and Gudden, Luciani, Tamburini, all showed that the decussation of the optic nerves is only partial. Goltz admitted, in 1876, that each cerebral hemisphere was related with both eyes in the dog. He argued, however, that the visual disturbance consequent on operation was hemiamblyopia, or cerebral weakening of vision, and not hemianopsia. Munk was slow to admit bilateral disturbances of vision from lesions of the occipital lobe. He long held that the dog differed in this respect from the monkey, though eventually he established the relations of either hemisphere with both retinæ in birds as well as mammals. Vitzow, Gudden, Ganser, followed in the same lines. Monakow (31) really determined the nature and conditions of bilateral homonymous hemianopsia. He showed by experiment that ablation of a cortical centre (Munk's visual sphere) reacted on the development of fibres and primary nervous centres connected with it, by the functional inactivity which it involved. His facts support the anatomical conclusions already quoted. The extension he gives to the region of cerebral vision in which lesions produce hemianopsia, is a reaction against the doctrines of Séguin, Nothnagel, and Henschen. The delimitation of this region is still, however, far from certain; as also the relation of the different parts of the occipital lobe to the various retinal segments. Monakow emphasises the importance of the primary optic centres interposed upon the optic path, such as the external geniculate body (in which the majority of the visual fibres terminate and lose their individuality). This fact is too often overlooked in schemata, which are constructed as if the projection bundles radiating over the visual sphere were the direct prolongation of the optic fillets.

This has important bearings upon the doctrine of retinal projection (*supra*). The anatomical conclusion appears to be that not the retinal fibres, but the fibres of the external geni-

culate body, are projected upon the occipital lobe. Monakow disallows the direct localised projection of impressions from the *macula lutea* on the cortex, his theory being that the macular fibres are distributed to all parts of the external geniculate body, and thus transmit their impressions to every part of the cortical centre. This would account for the partial preservation of central, or macular, vision in most cases of cortical hemianopsia.

Monakow's theory of the reciprocal relations, functional and trophic, that obtain between primary optic centres and occipital cortex is based upon secondary ascending and descending degenerations. These differ, histologically, and in their general physiological effects, according as the visual lesion is central or peripheral. After enucleation of the eye-ball in the dog, the ascending degenerations are sclerosis of the optic nerve, almost complete degeneration of the optic tract, slight diminution of volume in the motor nerve to muscles of the eye (oculo-motor, pathetic, external oculo-motor), general reduction of number of nerve cells, especially in the nucleus of the common oculo-motor. In dogs, cats, and man, the anterior quadrigeminal tubercles are little degenerated, while the external geniculate bodies are considerably atrophied—showing that most of the retinal fibres are closely connected with this body.

The same ascending atrophy was noted by Tomaschewski in the case of a child who became blind and deaf at the age of two, in consequence of meningitis. The ocular bulb atrophied from inflammation, and when the child died at the age of eight, the optic nerves, chiasma, tract, quadrigeminal tubercles, occipital lobe, angular gyrus, &c., were all atrophied.

Retrograde Degeneration.

Along with Wallerian degeneration, which attacks the peripheral end of a nerve separated from its trophic centre, there exists a retrograde degeneration, a "cellulipetal" alteration of the central end, propagated from the point of section to the trophic centre, *i.e.*, mother-cell of the nerve; and even in certain cases passing this centre to invade secondarily the following neuron. Marinesco (27) has observed a degeneration, varying in degree, in the central end of the peripheral nerves of amputated limbs. This involves the myeline and axis-cylinder, and determines in places a complete atrophy of the nerves, with proliferation of the interstitial tissue. Anatomically there is no essential difference between this process and that of Wallerian

degeneration at the peripheral end: it appears later, and advances more slowly; it is more rapid in young children than in adults.

Why do the nerves of the stump, and even cells in the anterior cornu, degenerate after amputation, seeing they are still in relation with their trophic centres, the spinal ganglia? The trophic centres themselves must suffer from the lesions of their nervous processes; the protoplasmic and axis-cylinder prolongations of a nerve-cell, with their terminal arborisations and collateral branches, are as much part of the cell as the limbs are part of the body of an individual; unity of the cell and its axis-cylinder is complete—a neuron is an individual. Golgi, Forel, Monakow, v. Gehuchten agree that lesion to any part of the neuron must bring about, first, the rapid destruction of that part of the nerve; and then the slower, but inevitable, death of the central portion of the neuron. The inter-annular segments of the nerves ("adipose cells traversed by the axis-cylinder of the nerve cell," Durante) may constitute proper sources of nutrition, rendering the nerve, in some measure, independent of the parent cell. We do not, indeed, know whether the nerve derives its sustenance directly from the cell, or whether it borrows from local exchanges along its course, the nerve cell merely regulating the processes of assimilation. Undoubtedly, the nerve is subject to local influences from its environment, *e.g.*, effect of chemical agents in circulation. Renaut (Lyon) strongly urges the local reaction of a nerve independent of the ganglion cell. At the same time, the axis-cylinder must always be regarded as an integral part of the parent cell.

Marinesco refers the trophic functions of the nerve-cell, and their ability to nourish and preserve the structure of the neuron, to the continuity of peripheral excitations (thus reconciling functional inactivity and trophic influence in the neuron; Türck, Bouchard). The effects of interference of nutrition after amputation may extend to the cells of the anterior cornu, and thence to the motor nerves and the muscles supplied by them (*atrophie musculaire dégénérative*). Cerebral muscular atrophy may also be referred to interruption of functional excitation, and the hypothesis of a special cerebral trophic centre eliminated.

Retrograde degeneration is found along the sensory path of the optic nerve, as in the spinal nerves. Lesions of the occipital lobe, or external geniculate body, induce retrograde or cellulipetal degeneration in the neurons of the optic path, which may extend

as far as the tract and optic nerve, although each such neuron has its trophic centre in its own mother-cell. The external geniculate body and pulvinar degenerate after lesion of the visual area, *i.e.*, have no autonomous visual activity, or reflex activity—such as attaches to the optic lobes of birds; and Monakow believes that each segment of the thalamus is in exact functional and trophic relation with its own section of the cortex. The characteristics of cellulipetal degeneration are a more or less pronounced atrophy of the myeline, followed by its total disappearance, which leaves the axis-cylinders bare, till they, too, perish. Turning to clinical evidence, Moeli has shown that in adults, affections of the occipital lobe determine descending lesions that may be followed into the nerve cells of the retina. He cites two cases of fœtal degeneration (porencephalia, internal hydrocephalia), due to lesion of the occipital lobe; the degeneration extended beyond the chiasma to the optic nerves. So, too, in another case: atheromatous softening of both occipital lobes in a man of 44, induced retrograde atrophy of the visual fibres and sub-cortical ganglia down to the optic nerve. Wilbrand, Henschen, and others found similar evidence for secondary descending degeneration. Nor does this stop at the optic nerve. The trophic disturbance of the basal ganglia affects the terminal arborisations of the opticus, and the descending alteration proceeds to the ganglion cells of the deep layers of the retina, and thence even to the rods and cones—the cellular prolongations in all cases being first to disappear.

M. Soury concludes from all these facts relating to the cortical localisation of bilateral, homonymous hemianopsia, that Ferrier is mistaken, and Munk correct, in their respective views concerning the cerebral centre of vision. Munk attributes the transitory hemianopsia, consequent on the extirpation of the angular gyrus, to the inflammatory reaction of the occipital lobe, and more especially to the compression (not destructive lesion) of the optic radiations of Gratiolet and Wernicke, which pass under the angular gyrus and inferior parietal lobe on their way to the cortex from the external geniculate bodies. It is thus that all lesions of the angular gyrus, &c., usually reach the projection bundle of the visual fibres. Should this be destroyed, and the message thus interrupted from the last sub-cortical station to the occipital cortex, nothing can avert absolute cortical, hemianopic blindness in the two halves of the retinæ implicated. Séguin (38), Forel, Nothnagel concur in locating the seat of vision, the optic field of perception, in the cuneus and first occipital gyrus. Unilateral lesion of these parts

produces hemianopsia, bilateral lesion produces total blindness.

Hemiopic Homonymous Hallucinations.

Tamburini defines hallucinations as a kind of epilepsy of the sensory centres. Visual hallucinations in the obliterated half of the field of vision may often be observed in hemianopsia of cortical origin. This is an extra phenomenon, super-added on the destructive lesion that produces partial cecity in both eyes, and due to an irritative lesion of the occipital cortex. Irritative processes resulting in hallucinations are frequently the precursor of destructive occipital lesions. Such visual hallucinations may even serve to determine the precise point of the cerebral cortex that is the seat of the lesion, *i.e.*, the hallucinatory alterations in the field of vision may be projected back upon the corresponding region of the "cortical retina." Soury does not accept the restriction of this irritative lesion to the calcarine surface of the occipital lobe (Lamy, *cf.*). He holds that the associations adequate to realise complex hallucinations must extend over much wider regions, probably over the convexity of the occipital lobe. He queries the possibility of unilateral hallucinations, and holds that just as a unilateral destructive lesion of the occipital lobe determines a homonymous bilateral hemianopsia, which produces partial cecity in both corresponding halves of the visual field, so a unilateral irritative lesion of the same region must determine a bilateral homonymous hallucination, which partially affects the visual fields of both eyes. Clinical evidence is quoted from Peterson, Colman, Hun, and Pick.

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(1) *Beitrag zum Studium der Medulla Oblongata, &c.* VON RAMÓN Y CAJAL. Translated by JOH. BRESLER, Leipsig, 1896.

(2) *Untersuchungen über die topographischen Beziehungen zwischen Retina, Opticus und gekreuztem Tractus Opticus beim Kaninchen.* PICK & HERRENHEISER, Halle, 1895.

(1) This is a *brochure* of about 140 pages, giving in a readable form the work of R. y Cajal upon the minute anatomy of the medulla oblongata, the pons Varolii, cerebellum, and origin of the cranial nerves. There is little to note that is not by now well known to most workers upon the anatomy and physiology of this region—facts which are gradually finding their way into the text-books, and which have already done much to explain many of the curious phenomena brought out by disease. The book is well illustrated, and there is an ample bibliography at the end.

(2) This is the record of an experimental research, performed with a view to trace the several bundles of fibres which constitute the optic nerve, optic chiasma and optic tract. A wound was made in the retina by means of a galvano-caustic needle, the operator being guided by the aid of the ophthalmoscope. After a limited time the animals were killed, and the nerves examined by the Marchi method. The following is a summary of the results: The fibres maintain the same relative position thoughout the optic nerve and tract. In the chiasma, the decussation takes place in a regular order, the innermost bundles of the nerve crossing first, and occupying an outer position in the tract, the lower bundles of the nerve occupying the lower part of the tract, and the outer bundles occupying the inner part.

It is probable (although there was a technical difficulty in the operation) that the uppermost bundles occupy the upper part of the tract. The decussation of the fibres is only in a transverse direction, there being no alteration of the fibres in a vertical direction. The several segments of the nerve correspond in general to like segments of the retina.

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